THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES.

DECEMBER, 1901.

PENETRATING GUNSHOT WOUNDS OF THE ABDOMEN.[1]

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Gentlemen: I have the opportunity of showing you this morning a case which illustrates a very dangerous and fatal class of injuries, and one which is not often available for clinical purposes, namely, a penetrating gunshot wound of the abdomen. Such wounds are among the most serious conditions that a surgeon is called upon to treat. Many such cases are of necessity fatal from their inception, owing to the irreparable damage inflicted by the bullet, and no amount of surgical skill will overcome the injuries received. In other cases, however, we have a fair opportunity for a successful issue, and in these instances failure or success will largely depend upon the surgical methods pursued. I therefore propose to give you a description of this man's injuries, the treatment we have adopted, and then discuss with you some of the important surgical points in this class of cases.

The patient, a well-developed negro laborer, aged twenty-seven years, was admitted to the Pennsylvania Hospital, September 7, 1901, at noon. Fifteen minutes before admission he was twice shot with a 32-calibre revolver. The first bullet caused a superficial wound of the left thigh, but the second struck him in the back while he was running away from his antagonist. The estimated distance at which this shot took effect was twenty feet. I saw the patient two and a half hours after the injury was received. He was then in a highly excited condition, in part, perhaps, due to the fact that his ante-mortem statement had just been taken by a magistrate, and the necessary legal procedures had terrified him. He complained of intense, cramp-like pains in the abdomen, which made him cry out every few minutes. He was moderately shocked,

[1] Clinical lecture delivered at the Pennsylvania Hospital, October 12, 1901.

with a rapid pulse, and labored respiration which was almost entirely costal. The wound of entrance was three inches to the right of the fifth lumbar spine and on a level with the crest of the ilium, and a probe refused to pass beyond the latissimus dorsi aponeurosis. There was no wound of exit. The abdomen was universally tender, distended, and resistant, tympanitic in front and dull in the flanks. Liver dulness in front reached to the costal border. The man was etherized, placed on his face, and the skin of the back carefully disinfected. As the probe still refused to follow the track of the bullet, the skin wound was enlarged sufficiently to see where the missile had separated the fibres of the aponeurosis. These fibres were divided, and a finger was carried along the track of the bullet to the crest of the ilium, where two or three splinters of bone were encountered. The bullet had then passed beyond the reach of the finger. Having assured ourselves that the missile had penetrated the peritoneal cavity from behind, a rubber drainage-tube was inserted in this track and the incision closed. The patient was then turned on his back, the skin of the abdomen carefully disinfected, and a six-inch incision made in the median line, the centre of which was the umbilicus. On opening the abdomen fluid blood immediately gushed out; the quantity was estimated at six pints. The whole of the small intestine was hastily drawn out of the abdominal cavity to the left of the incision, and surrounded with hot, wet gauze. In doing this a perforation of the small gut was seen. Its position was noted and the place covered with gauze, to prevent further extravasation of fecal material. The abdominal cavity was then cleared of blood by scooping it out with the hand and with gauze sponges, and it was seen that the bullet had perforated the upper portion of the mesentery, severing one of its large vessels. As it emerged from the mesentery it had torn an opening one inch long in the transverse colon. The hemorrhage was readily controlled by suture, the wound of the colon repaired, and the perforation of the small gut closed with silk. No further damage was found. Twenty pints of hot salt solution were used in washing out the abdominal cavity and the surface of the intestines, a glass drainage-tube was inserted, and the wound hastily closed with through-and-through silkworm-gut sutures. During the operation the patient's pulse had become very rapid and feeble, and an assistant opened the median cephalic vein and introduced five pints of hot normal salt solution. As the bullet had almost certainly lodged in the anterior abdominal wall, and was out of harm's way, no search was made for it, the patient's condition being so precarious as not to warrant any further interference.

Immediately after operation the patient's temperature was 98.4° F., pulse 160, and respiration 60. Eight hours later the temperature had risen to 101° F., while the pulse and respiration had dropped to 124 and 44 respectively. He vomited twice while coming out from the effects of ether. During the first twenty-four hours he received medication by hypodermic only, and nothing was administered by the mouth.

The treatment consisted of one-thirtieth of a grain of strychnine every second hour, and as the pulse increased in volume and decreased in frequency, this was gradually reduced in amount. At the end of twenty-four hours one-sixth of a grain of calomel, with a mouthful of water, was given hourly for six doses. This was followed by an enema, which proved very effectual. As soon as the bowels were opened he was placed

on peptonized milk, and the diet gradually increased from day to day. In the meantime the drainage-tube had been very carefully kept dry by having the accumulated fluid sucked up with a syringe. At first this had to be done every few minutes, owing to the large amount of salt solution which was left in the abdominal cavity, but at the end of six or eight hours the amount of fluid had so decreased that the drainage-tube was cleaned only once every hour or hour and a half. At the end of forty-eight hours the tube was entirely dry, and it was removed. At the time he received his injury the patient was suffering from a mild bronchitis, and during the convalescence he had a severe cough, with free expectoration. This was partly controlled by heroin, but the paroxysms of coughing everted the edges of the wound in three places, although none of the stitches gave way. The sutures were removed on the eleventh day, at which time also the rubber drainage-tube in the back was taken out, and the patient was out of bed on the twenty-second day. Owing to the fact that the X-ray apparatus in the hospital is not at present in a working condition, we have not been able to take a radiograph for the localization of the bullet in the anterior abdominal wall, but as soon as we can have a picture for a guide we will remove the bullet.

In discussing the subject of penetrating gunshot wounds of the abdomen with you, it is necessary that we consider in detail a number of important points.

1. *General Considerations.* The patient's temperature, pulse, and respiration should be taken as soon as possible, as it will give a fair idea of his general condition. While this is being done it is well to pass a catheter and withdraw the urine, for if it be tinged with blood we will know that some portion of the genito-urinary tract has been injured—either the kidney, ureter, or bladder. Next, when possible, we will get a history from the man of the position in which he was at the time he was shot, and from this estimate the probable course the bullet has taken in traversing the abdomen. In doing this you will naturally picture to yourself the organs that are likely to be wounded. Of course, this will be more or less of an assumption on your part, for bullets are so readily deflected that the direction of the wound of entrance does not always give us a clue to the further passage of the bullet through the body.

2. The question will then come up, Shall we or shall we not operate? Up to a few years ago a gunshot wound which opened any part of the alimentary tract was considered fatal unless operation was undertaken, and even then almost all cases died. At that time all gunshot wounds were alike, in that they were dirty, usually infected, lacerated wounds made by a comparatively large soft lead bullet of slow velocity. The bullet, in addition, was usually covered with grease. To-day we have a gunshot wound of very different character, made by a clean, small, highly polished steel bullet of high velocity. When such a missile has attained its true flight, it leaves a clean, sharp-cut wound, separating

the tissues as a wedge rather than tearing its way through. Penetrating abdominal wounds made by a lead bullet I believe are as fatal to-day as ever they were, whenever expectant treatment is pursued; but military statistics of field operations show that the wounds inflicted by the modern weapon have proved much less fatal under expectant treatment than when operation has been undertaken. We can scarcely wonder at this when we remember the circumstances under which operations are performed after battle in field hospitals, where the medical department has an immense work to do with scanty supplies and few assistants; but if such wounds presented themselves in a city hospital, with all the modern conveniences for aseptic surgery, would any of us hesitate between expectant and operative treatment? I should say no. Wherever we can with safety open an abdomen, I believe that every penetrating gunshot wound demands operation, no matter what the character of the bullet may be which has inflicted it; but where our surroundings are such that we cannot do a clean operation, expectant treatment will yield better results, especially if the wound is made by a modern bullet.

3. *Shock.* When shock is profound, should we wait for reaction to take place? Shock in such cases is almost invariably associated with hemorrhage, and its severity is almost directly proportional to the amount of blood lost. Occasionally, however, it is profound where hemorrhage has been slight. Here we will frequently find that the patient is of a highly nervous temperament, the type of individual who would be nauseated or faint at the sight of blood. Occasionally, also, profound shock may be due to very extensive injury to the hollow viscera, or, perhaps, contusion of some larger nerve trunks. Under such circumstances, what advantage can we expect to derive by delaying operation? To my mind, none. While an anæsthetic in the presence of profound shock is, of course, dangerous, this increased danger in no way counterbalances the benefits derived from its use. It will permit us to get at and control the cause of shock, by preventing further hemorrhage or greater soiling of the peritoneum; it enables us to repair the injuries inflicted and to reduce the dangers of septic infection to a minimum, and at the same time it permits the use of the two most valuable remedies we have for combating shock, namely, intravenous injection and douching the abdominal cavity with hot salt solution. I would therefore urge, that if operation is to be undertaken, it should be done immediately, as every half-hour of delay proportionally decreases our chance of success.

4. *Examination of the Wound of Entrance or Exit to Prove Penetration.* When the wound of entrance or exit does not give a positive sign of penetration, as the protrusion of some part of omentum or escape of fecal material, it must be carefully enlarged until the peritoneum is

reached, when sight or touch will tell of penetration. We should never take for granted that the abdominal cavity has been invaded from the apparent course the bullet has taken, nor from the local signs that may be present, such as tenderness, distention, muscular rigidity, shock, etc. All these signs may be present without an opening into the peritoneum. Four or five years ago I saw a very stout woman who was shot with a revolver. The wound of entrance was on a level with the left anterior superior spine of the ilium, and about two and a half inches to its inner side. The apparent course taken by the bullet was directly inward, and there was no wound of exit. The patient was considerably shocked, slightly tympanitic and distended, with rigidity of the left rectus, and marked local tenderness. I was confident the abdominal cavity had been opened, but proceeded to assure myself of this by exploring the wound. It was then proved that the bullet had ranged downward, and had penetrated to but not opened the peritoneum, passed under Poupart's ligament, and lodged in the muscle of the thigh. The woman was very stout with a pendulous abdomen, and as she reclined on her back the wound of entrance was on a level with the anterior superior spine of the ilium; but in a standing position, the position in which she was shot, the wound undoubtedly assumed a relatively lower position. You will therefore see that the external signs of penetration are not always to be relied upon, and that assured proof, either by sight or touch, is indispensable.

5. *Cleansing the Skin of the Patient.* I wish to call your attention to the importance of thoroughly disinfecting the skin of the patient previous to opening the abdominal cavity. As a rule, this cannot be well done until the patient is under an anæsthetic, owing to abdominal pain and tenderness, and at this time we are apt to do it hastily and ineffectually, when the patient's condition is precarious, in our desire to shorten the time on the operating-table. Time spent in cleansing a large area of skin around our proposed incision is well expended, and if our patient's condition is so grave that we dare not spare this time, operation should not be undertaken. Experience is the great teacher, and when one can learn from the mistakes of others it is more profitable than learning from one's own errors. I am therefore going to make a confession, which I trust will be instructive. Several years ago, before the days of normal salt solution and intravenous injection, when speedy operations seemed the most successful, I had a case of gunshot wound of the liver. The wound of entrance was in the back, the bullet having passed through the pleura, diaphragm, and whole thickness of the liver, and lodged under a rib in front. The man was profoundly shocked from loss of blood. I enlarged the opening in the back and packed the liver wound as thoroughly as possible from this position, and then, turning the patient over, made a curved incision along the border of the ribs, and completed the packing of the liver from the front. The patient's con-

dition was so serious that I did not spend over three or four minutes in cleansing the skin of the abdomen. He was dying from hemorrhage, and the indication seemed to be to stop the bleeding as soon as possible. After operation he reacted well, but died on the fifth day from septic peritonitis. A post-mortem examination revealed the wounded liver in excellent condition; no further hemorrhage had taken place, and the bullet track was filled with organizing sterile clot. Infection had started from the skin of the abdominal incision, and was traced, layer by layer, through the abdominal wall to the peritoneum, where it had become diffuse. In trying to save the man from a death by hemorrhage, I had unfortunately condemned him to one from peritonitis. Had ten or twelve minutes been spent in disinfecting the surrounding surface of the abdomen, infection would not have taken place, and the man would have undoubtedly recovered. Let us look at the question in another way. I am in the habit of spending fifteen minutes or more in preparing my hands before opening the abdominal cavity. Why should not the same careful preparation be given to the abdomen of the patient, when it is most probable that some portion of the peritoneal surface will come in contact with it before the operation is concluded?

6. *The Incision.* Penetration having been ascertained, where shall we make the incision for exploring the abdominal contents? This should depend upon two factors—the position of entry and the course the bullet has taken. If the missile has penetrated outside of the semilunar lines, and at a right angle to the anterior abdominal plane, I would open the abdomen in the semilunar line, rather than enlarge the bullet wound sufficiently to make a careful search of the abdominal organs. If it has entered in the same plane, but inside the semilunar line, I prefer a median incision, unless it is in the region of the liver, when a curved incision below the border of the ribs gives the best exposure. When the bullet has entered the flank or back, and ranged across or diagonally across the abdomen, I should always prefer a median incision, except when the liver is probably the only organ penetrated. An incision through the median line or semilunar line saves time, as it can be enlarged and closed more quickly than when several layers of abdominal muscle have been opened; it permits of a more extensive and thorough examination of the abdominal contents, and also of a better cleansing technique before closing the abdomen.

7. After the abdomen is opened, the conditions which present themselves at this time will determine your further steps in the operation. If a large quantity of blood is encountered, the first indication will be to stop the hemorrhage. To do this the small intestine must be withdrawn from the abdominal cavity, and surrounded and covered with hot, wet gauze, the blood then removed from the peritoneum, and the source of hemorrhage searched for. When the solid organs are not in-

volved in the injury, the most frequent source of hemorrhage is probably the mesentery, and until the small intestine has been withdrawn from the abdominal cavity, we have no opportunity of making a careful examination of the mesentery. If the vessel causing the hemorrhage can be ligated or sutured, this should be done at once. When the hemorrhage is taking place from the solid organs, as the liver, spleen, or kidney, we should first attempt to control it by packing the damaged tissue with gauze, but in the case of the spleen or kidney this will not often prove successful, and the removal of the organ will then be demanded. Hemorrhage having been controlled, we will next search for perforations in the alimentary tract. It is well here to start with a definite system, and, beginning with the stomach, follow the small intestine throughout its windings, and then go to the large intestine. As each perforation is found it is immediately closed with the Lembert sutures, catgut or silk being employed for this.

The practice of inflating the intestine by means of hydrogen introduced in the rectum, for the purpose of demonstrating the points of perforation in the alimentary tract, is, to my mind, distinctly bad surgery. It wastes time, it increases fecal extravasation, and so distends the intestines that it may be impossible to return them to the abdominal cavity until they have been emptied of the gas. If your abdominal incision is of proper size and your search systematic, perforations of the bowel will not escape you. Sometimes the intestine will be so greatly damaged that a simple closure of the opening would cause stricture or sharp kinking of the gut, leading to a later obstruction of the bowel. This is particularly liable to occur in marginal wounds of the mesenteric border. Under such circumstances a resection of the gut is necessary, but before proceeding to it, examine the intestine for two or three feet above and below, to see that there are no further wounds of this severe type. If you find one or more, it will be safer to include all the damaged area in a single resection, provided you do not remove more than thirty or forty inches of gut, rather than make two or more resections within a short distance of each other. If the anterior wall of the stomach is perforated, close it in the same manner as a wound of the intestine, and then break through the gastrocolic omentum to examine the posterior wall for a wound. From the anterior wound no attempt should be made to locate an injury to the posterior wall, for such a procedure would necessitate the soiling of the hands of the operator, and also endanger the peritoneum by forcing out the stomach contents. While you are carrying on these necessary procedures, it must be the duty of an assistant to keep the bowel exposed outside the abdominal cavity moist and warm, by repeatedly renewing the hot, wet gauze surrounding it. Having repaired all the damage, we flush the abdominal cavity liberally with hot salt solution, and also carefully douche

the intestines which have been brought outside of the abdomen with the same. The intestines are then returned to the abdominal cavity, and as much salt solution as possible allowed to remain within the peritoneum.

8. *Search for the Bullet.* If the bullet is not encountered when we are examining the abdominal organs for wounds, it presumably has passed outside of the peritoneal cavity and lodged in muscular tissue or bone. In such a position it is practically out of harm's way, and when the patient's condition is serious no time should be lost in a further search for it. At some later period, when convalescence is established, the X-ray will easily show us where the bullet has lodged, and it may then be removed with safety to the patient.

9. *Drainage.* In operating for gunshot wounds in other portions of the body, it has always been my practice to treat the track of the bullet as septic until it has proved itself otherwise, and I have always drained such wounds. If, in addition to a septic bullet, the alimentary canal has been opened, the indications for drainage become doubly imperative. When the bullet has penetrated the lesser peritoneal cavity, as in wounds through the stomach, part of the duodenum, and the upper portion of the transverse colon, I believe that a counter opening should be made in the flank, in order that this cavity may be perfectly drained. The danger of infecting the peritoneum from outside sources through the drainage-tube is certainly very slight when compared with the dangers arising from the presence of septic material in such a closed cavity.

10. *Closing the Wound.* As the patient will generally be in a precarious condition, it is well not to spend much time in closing the abdominal wound, and for this reason a through-and-through suture of silkworm-gut is probably the best we can use, although where time is no object a layer suture will be more apt to prevent future hernia.

11. *After-treatment.* For the first twenty-four hours the tube has to be very carefully attended to and thoroughly cleaned as often as it fills with fluid. During this time nothing is given to the patient by the mouth, and any drugs which we find it necessary to administer should be given hypodermically. At the end of twenty-four hours, except in wounds of the stomach, it is my custom to administer calomel, with a small quantity of water, and to have the bowels moved as soon as possible, either by enema or by the administration of repeated small doses of salts. If the patient's condition is such that food is imperative, we can use the lower bowel for this purpose. As soon as the bowels have moved, small amounts of nourishment may be given by the mouth at frequent intervals, and if no untoward symptoms present themselves, like distention, pain, etc., the amount of nourishment may be rapidly increased. When the drainage-tube has ceased to fill it must be withdrawn. The complication most to be feared is, of course, obstruction of the bowels, and

for combating this we have to use energetic cathartics, with frequently repeated enemata. For the rest, the after-treatment is similar to that followed in all cases of laparotomy.

In closing, let me recapitulate briefly some of the most important principles in the treatment of this grave lesion:

1. Remove the patient at once to the nearest place where a clean operation may be undertaken.

2. Assure yourself positively that penetration has taken place.

3. Having demonstrated this fact, always open the abdomen and search for injuries, and make this search systematic.

4. Never wait for symptoms to tell you that profuse hemorrhage or intestinal perforation has taken place, for by that time operation will usually be useless.

Lastly, remember that cleanliness and thoroughness must never be sacrificed to speed. These two are absolute essentials of success, while speed is no longer an important factor since the introduction of normal salt solution.

REPORT OF A CASE OF SPORADIC TRICHINOSIS.

By John Chalmers Da Costa, M.D.,
of Philadelphia,

and

Rae Shepard Dorsett, M.D.,
of Philadelphia.

The following report presents many interesting and some important points.

The patient, T. M., was admitted into St. Joseph's Hospital, March 6, 1901. He is white, unmarried, and twenty years of age. His mother died of phthisis. His father and only brother are living and well. He had measles and whooping-cough in childhood, but never suffered from any other serious sickness. Never uses alcohol, and smokes moderately. The young man says that the right lower extremity has always been distinctly larger than the left, and his father confirms this statement. When thirteen years of age he left school and began work as a delivery clerk for a dry-goods house. He followed this occupation until September, 1900, when he became an assistant to a milk dealer. He did not suffer at any time during the summer with gastro-intestinal disturbance. Early in August, 1900, he took a holiday and started for Atlantic City upon a bicycle. On the meadows about Atlantic City he dismounted from his wheel and stood for a time to wait for the passage of a railroad train across the path. While standing, another bicyclist ran into his machine, and the pedal of the second bicycle struck T. M. upon the calf of the right leg. The blow caused much pain and disablement, but did not break the skin. After a wait of half an hour

he proceeded to Atlantic City upon the rear seat of a tandem bicycle. On reaching his destination he went to bed for several hours. At the termination of the rest, in spite of the fact that the calf was swollen and somewhat painful, he arose from bed, went out, put on a bathing suit, and went in to bathe. He states that while walking along the beach someone called his attention to the fact that blood was running down his leg. The blood flowed freely for ten minutes, and did not come from a cut, but "flowed through the pores." This remarkable statement he iterates and reiterates. He returned to the city on the afternoon train. The leg was somewhat sore for a few days, but he went back to his work. The swelling diminished, but did not entirely disappear. For almost four months he suffered no inconvenience with the leg.

In the early part of January, 1901, he noticed that the swelling was increasing. During January and February the size of the calf of his right leg increased very much, and during February he began to suffer from a sense of soreness on walking and from attacks of cramp-like pain at night. If the leg were kept in a flexed position for some time it could be extended with difficulty, and the effort was productive of acute, crampy pain.

Examination on admission: The calf of the right leg is much enlarged, and the gastrocnemius muscle from origin to insertion is clearly traced even when in repose. When he rises on the toes the muscle bellies out in the most marked manner. Muscular movements produce soreness, but no acute pain. While at rest there is no spontaneous pain during the day, but he has cramps at night. There is no tenderness of the muscle and no œdema of the skin. The movements are not notably interfered with; he is clumsy, but is not lame. The swelling is of uniform consistence, does not pulsate, and feels like a mass of thick rubber. The cutaneous veins are much dilated. The groups of muscular fibres can be distinctly outlined under the skin, as though no fascia intervened. The thigh is slightly larger than the opposite thigh, and the foot is somewhat larger than its fellow, but in neither the thigh nor foot is there pain or tenderness. The temperature is normal, the bowels are regular, his appetite and digestion are good, the urinary examination is negative; the lungs and heart are normal. He complains of nothing but the swollen calf.

During the next three weeks the temperature remained normal. An examination of the blood showed about 12,000 leucocytes, 4,300,000 erythrocytes, 60 per cent. of hæmoglobin, and no increase of eosinophiles.

It was observed, however, that the thigh was undoubtedly increasing in size, and so were the flexor muscles of the sole of the foot. In the thigh both extensors and flexors were enlarged, but there was neither pain nor tenderness. In the foot and leg the flexors alone were obviously involved. The pain continued as before in the leg, but there was no tenderness, and the foot was free from any uneasiness.

We were much puzzled as to the diagnosis, and invited Dr. Dercum to discuss the case with us. The alleged bleeding through an unbroken skin was eliminated from any relation to the diagnosis. We believed that it was due to a small rupture in a varicose vein.

At first we had thought it possible that the injury had partly ruptured the muscular sheath, the rupture having subsequently extended

and the entire muscle having emerged as a hernia. This view, which never seemed very probable, because of the lack of marked functional impairment, and because of the fact that we were unable to feel the rigid margins of torn sheath, became quite untenable when the other muscles were found to be involved. It was certainly not pseudohypertrophic paralysis or sarcoma. There was obvious muscular hypertrophy and possibly myositis, and the entire absence of fever was against infective polymyositis. We determined to make an incision and remove a piece of the gastrocnemius muscle.

March 27th. An incision was made. The muscular sheath was found intact. A bit of muscle was removed. It contained some white spots visible to the naked eye. Dr. Rosenberger examined the piece of muscle

FIG. 1.

Section of gastrocnemius muscle. Heidenhain fixation, hæmatoxylin and picric acid. Photomicrograph; Leitz 8 mm. objective, Zeiss objective eye-piece. In the upper part of the field is shown a capsule containing oblique and transverse sections of a coiled trichinæ. In the centre of the field is a calcifying capsule, near the margin of which is a collection of lymphoid cells. The parasite in the lower left quadrant of the field is beginning to disintegrate.

microscopically and found it swarming with embryo trichinæ. (See Fig. 1.)

Subsequent to receiving the pathologist's report renewed questioning of the patient developed the statements that he had never eaten pork until going to live with the milk dealer, but after this time (September 1st) he had eaten pork, on an average, four times a week. He stated that he never ate raw ham or sausage—only cooked pork.

Figs. 2, 3, and 4 show photographs of the patient taken April 1st.

Repeated blood examinations made by Dr. Rosenberger and Dr. John C. Da Costa, Jr., showed a gradual advance in the number of leucocytes. For instance: 12,600 on April 3d, 17,600 on April 11th,

21,500 on May 4th. There has at no time been an increase in eosinophiles noted. The blood report on May 4th is as follows: 69 per cent. polynuclear, 3.5 per cent. eosinophiles, 22.5 per cent. lymphocytes, 5 per cent. hyalin; erythrocytes, 5,212,800; leucocytes, 21,500; hæmoglobin, 75 per cent.

Up to the present time (June 27th) the right lower extremity has continued to increase in size. Recently the left lower extremity has begun to enlarge, but is not the seat of pain or tenderness.

April 5th. The measurements were as follows: Maximum circumference of right leg, 15 inches; maximum circumference of left leg, 13

FIG. 2.

inches; circumference of right thigh 5½ inches above upper margin of patella, 18 inches; circumference of left thigh at the same level, 15½ inches.

22*d.* Measurements: Of each thigh, same as April 5th; of right leg, one-half inch greater.

27*th.* A bit of the left deltoid muscle was removed, and was found to be swarming with trichinæ, although the muscle was not painful and not obviously enlarged.

May 4th. Right leg, 15¾ inches; left leg, 13½ inches; right thigh, 18¾ inches; left thigh, 15¾ inches.

Patient returned home the 1st of May, and whereas the swelling has not diminished in the slightest, he feels very comfortable, does not suffer to any degree from cramp, walks freely and easily, and regards himself as in excellent health except the swelling of his right lower extremity.

FIG. 3.

FIG. 4.

The particular features of interest in this case are that a traumatism preceded the apparent onset of the muscular trouble; that the pain in the muscle was slight; that tenderness of the muscle and œdema of the skin were absent; that no antecedent gastro-intestinal attack occurred; that fever was absent; that, in spite of wide-spread invasion, some diseased muscles (the deltoid) did not appear to be swollen and were not tender or painful, and other invaded muscles (the opposite lower extremities) were enlarged, but gave no evidence of pain; finally, that eosinophilia was never noted.

The possible relation of the traumatism to the subsequent condition is a point of decided interest. The injury was received early in August.

Some swelling remained, and early in January it began to increase. Can we suppose that the seat of injury constituted a point of least resistance toward the larvæ trichinæ just as an injury often does toward micro-organisms. The fact that pork had never been eaten until September seems to render untenable the suggestion that trichinæ encapsulated in the muscle were set free by the injury, even if such a happening were possible. There is a third view: that is, that the injury had nothing whatever to do with the origin in the calf, the association being casual and not causal; but it is at least peculiar that the disease should begin in such an unusual situation as the calf, and that in this very situation there should have been severe damage inflicted a few months before.

The absence of eosinophilia is proof that this supposed diagnostic test is not conclusive. It may be usually present, but is certainly sometimes absent.

The fact that muscles can contain numerous trichinæ without obvious signs being engendered has been observed in other cases.

The conclusion is that in a doubtful case of muscle enlargement we must think of trichinosis as a cause, and must confirm or set aside this diagnosis by incision, removal of a piece of muscle, and examination of the piece removed by means of a microscope.

The report of Dr. Randal C. Rosenberger is appended.

Report upon a Case of Trichinosis, with Remarks on (1) Trichinosis and (2) Eosinophilia.[1]

Pathological Report. The specimen consists of a small, irregular cube of muscular tissue, each side measuring 2 cm.

A small portion was fixed in 1 per cent. osmic acid, dehydrated, and embedded in paraffin; the remainder of the specimen was fixed in Heidenhain's solution, dehydrated, and embedded in paraffin.

Sections were cut and examined in the unstained condition (those fixed in osmic acid); others were stained with hæmatoxylin and picric acid, hæmatoxylin and eosin, and with Van Gieson's mixture.

Upon microscopical examination the specimen is seen to be made up of transverse, longitudinal, and oblique sections of striated muscle.

Some fibres show cloudy swelling (the striations are not demonstrable), while others are irregular and wavy. Here and there are small areas in which fragmented, split fibres are seen. The muscle nuclei are for the most part normal, but in some areas a notable proliferation is clearly in progress. Small and large recent hemorrhages are seen, mostly between bundles of fibres, while one or two extend into and between the individual fibres. Increase in the amount of connective tissue is evident in some areas, and large numbers of small round-cells and spindle-shaped cells, with rod and irregular wavy nuclei, are present.

Between and sometimes in the muscle fibres encapsulated parasites are seen. In some capsules only transverse sections of the parasite can be seen; while in others longitudinal; and in still others the whole

[1] Read before the Pathological Society of Philadelphia by Dr. Randal C. Rosenberger.

parasite can be observed arranged in a spiral or coiled condition. As many as eight parasites can be seen in one field with a two-thirds inch objective and one-inch eye-piece (Bausch and Lomb).

The capsules contain, besides the parasites, a certain quantity of granular material, most evident at each end, a few small, oval, apparently vacuolated cells, and an occasional small round-cell.

Around the capsule is seen in many areas a marked round-cell accumulation, with some few spindle-shaped cells; other capsules show this change to only a slight degree.

A few capsules are devoid of parasites; the walls of such capsules are intensely thickened, and some contain vacuolated cells and granular detritus; the latter may be calcareous matter.

Mast cells are abundantly scattered throughout the specimen.

Sometimes three and four parasites are found within one capsule; in the sections studied in the case here reported but one parasite could be identified in any capsule

The following blood-counts were made:

1. April 3, 1901.

Hæmoglobin	48 per ct.
Erythrocytes	4,336,000
Leucocytes	12,680

Differential Count of Leucocytes.

Finely granular oxyphiles	65.5 per ct.
Coarsely granular oxyphiles (eosinophiles)	4.0 "
Lymphocytes	21.0 "
Hyalin	9.0 "
Basophiles	0 5 "

2. April 11, 1901.

Hæmoglobin	80 per ct.
Erythrocytes	4,275,000
Leucocytes	17,600

Differential Count of Leucocytes.

Finely granular oxyphiles	63.0 per ct.
Coarsely granular oxyphiles (eosinophiles)	2.5 "
Lymphocytes	27.5 "
Hyalin	7.0 "
Basophiles	0.0 "

3. April 21, 1901.

Hæmoglobin	72 per ct.
Erythrocytes	5,200,000
Leucocytes	20,000

Differential Count of Leucocytes.

Finely granular oxyphiles	68.5 per ct.
Coarsely granular oxyphiles (eosinophiles)	3.0 "
Lymphocytes	22.5 "
Hyalin	6.0 "

4. May 4, 1901.

Hæmoglobin	75 per ct.
Erythrocytes	5,212,000
Leucocytes	21,500

Differential Count of Leucocytes.

Finely granular oxyphiles	69.0 per ct.
Coarsely granular oxyphiles (eosinophiles)	3.5 "
Lymphocytes	22 5 "
Hyalin	5.0 "

The granules of the finely granular oxyphiles were generally markedly oxyphilic, but could not be taken for eosinophiles.

Remarks on Trichinosis. In 1822 Tiedemann, and in 1832 Hilton, noticed calcified cysts in the muscle. Leidy, in 1847, first found the trichina spiralis in pork.

In 1845 Owens discovered the trichina spiralis in a cadaver in the dissecting-room, and in 1848 the same parasite was found by Herbst.

It is well known that Zenker,[1] in 1860, was the first to call attention to the disease trichinosis, and almost at the same time Leuckhart confirmed the relationship of the parasite as a cause of the disease. In 1862 Friedreich[2] first diagnosticated the affection and experimentally determined the presence of the parasites in the living subject.

In experiments upon birds the trichinæ advanced in development in the intestine, but failed to invade the muscular system.

Embryo trichinæ are generally present in the intercostal muscles, muscles of the eye, larynx, and tongue, but can be found in almost any muscle of the body. In the case just reported the most severe infection seems to be localized in the gastrocnemius muscle, although sections of the deltoid also show the presence of the parasites.

Not only do the trichinæ encyst themselves in the voluntary muscles, but also in the panniculus adiposus and in the coats of the intestine. In the larger muscles the parasites are most numerous near their tendinous ends. The heart rarely contains trichinæ. The parasite possesses great powers of resistance to putrefaction, being found alive one hundred days after exposure; they survive three days' exposure to temperatures of 22° to 25° C. According to Perroncito,[3] the parasite resists a temperature of 48° to 50° C. for only a few minutes; other observers, however, claim that a temperature of 60°, 70°, or 80° C. is necessary to kill.

According to Roberts,[4] the muscle fibres involved are generally, upon microscopical examination, found to be more or less destroyed and the interstitial connective tissue increased.

Dr. Sutton,[5] harpooning a small mass of gastrocnemius muscle in a fatal case of trichinosis, found it swarming with trichinæ, "estimated at more than 100,000 to the square inch;" the parasites were in active motion, "coiling and uncoiling."

Tyson[6] mentions that the capsule may undergo fatty degeneration, which takes place at times early, but may be delayed for years. The worm-cyst lies usually with the long axis parallel to the muscle fibres.

A. M. Lewin[7] points out that trichinal myositis has its seat entirely in the muscle elements, whereas anthrax myositis is confined to the connective tissue. In Dr. Da Costa's case the increase in connective tissue seems to be between as well as in the muscle fibres.

R. Langerhans claims that trichinæ, together with their capsules, are subject to degenerative changes, and eventually reach a stage of elimination from their host. He further claims that recovery is never complete until the total elimination of both parasite and capsule takes place.

The oval cells seen within the capsules are, according to Heller,[8] "oval, vesicular-shaped muscle nuclei," formed by the thickened sarcolemma.

The œdema of the parts involved is said by Colberg[9] to be due to the obliteration of the capillaries of the muscles in which the trichinæ become embedded. Klob[10] suggests that the œdema may be produced by the lymphatic channels becoming plugged with the parasites, the lymph stream being thus obstructed. Hamilton[11] thinks that the œdema is of an inflammatory nature, due to the irritation caused by the parasites.

According to Sterling and Verco,[12] the capsule of the cyst is chitinous and lamellated, varying a good deal in thickness according to the part of the cyst examined and to its age, being always thickest at the poles and in the oldest cysts, least dense and most transparent in young cysts and about the equator. They also mention that as many as six or seven trichinæ may be found in a common capsule.

Pagenstecher, Chatin, Cerfontaine, and Askanazy[14] state that the female trichinæ penetrate into the intestinal villi and deposit their embryos in the lymph vessels, whence their migration occurs.

Remarks on Eosinophilia. The blood-counts here recorded do not show an unusual increase in the percentage of eosinophiles.

Brown[13] claims that there is constantly a relative increase in the eosinophiles, which may reach 68 per cent.

Eosinophilia is by no means pathognomonic of trichinosis, as the same condition is noticed in other processes.

Turk, in pneumonia, found a post-critical eosinophilia of 5.67 per cent.; after acute articular rheumatism, 9.37 per cent. Even after the injection of tuberculin eosinophilia may be noticed, and Zappert in several cases records 26.9 per cent. of eosinophiles.

Osler[15] in five cases of trichinosis (diagnosis confirmed by finding the parasites in the muscles) found quite a pronounced eosinophilia in each case. In the first case T. R. Brown (the clinical clerk) gives 37 per cent. eosinophiles. Up to the date of the patient's discharge the eosinophiles averaged 16.8 per cent. The second case, counted by Dr. Futcher, showed 44 per cent. eosinophiles. The third case, counted by Dr. Gwyn, showed 45 per cent. eosinophiles. The fourth case, counted by Dr. Thayer, showed 48 per cent. eosinophiles. The fifth case,

counted by Dr. Gwyn, showed from 22 to 36 per cent. eosinophiles, and after the "patient had been up and about for three weeks" the eosinophiles were more than 60 per cent.

Sears,[16] reporting a case of trichinosis, found in the first count 17.5 per cent, and in a second count 13 per cent. of eosinophiles. A well-marked urticarial eruption was present over the trunk and thighs.

In Lambert's and Brook's case of trichinosis, quoted by Ewing,[17] there was no evidence of transformation of neutrophiles into eosinophiles, although some of the eosinophile granules were unusually small.

Blumer reports a case in which there was 50 per cent. eosinophiles; a recovering case by Brook gave 83 per cent. Ewing claims that from 40 to 60 per cent. are usually found, but at some periods they may fall to 8 or 10 per cent. The eosinophilia is usually persistent, 15 per cent. remaining after four and a half months in a case reported by Stump, and 34 per cent. after five months in a case reported by Brown.

R. J. Patek[18] reports a case in which there were 30 per cent. eosinophiles, the trichina spiralis being found in a sample of sausage from which the case had its origin.

In infections by other animal parasites, as the anchylostoma duodenale, Muller and Rieder found 8.2 per cent. to 9.7 per cent. of eosinophiles. Zappert also noticed eosinophilia in the same condition.

Bucklers[19] found 16 per cent. eosinophiles in a case of oxyuris vermicularis; 19 per cent. in ascarides. Lichtenstein found 3.4 per cent. in a case of tænia mediocanellata, and 72 per cent. in a case of anchylostomiasis. He also reports 13.5 per cent. of eosinophiles in a case of anguillula stercoralis.

Grawitz regards the increase of eosinophiles in afebrile stages of malaria as of diagnostic import. Zappert, in a case of malaria one day after the last attack, found 20.34 per cent. eosinophiles.

Engel, in varicella, when the pustules were healed, found 16 per cent. eosinophiles. Six per cent. of eosinophiles has been observed by Wickery in von Jaksch's anæmia infantum.

Ehrlich claims that there is a marked eosinophilia in leukæmia, and further states that this is a pathognomonic sign of the disease.

Reinbach found an increase in the number of eosinophiles in the blood of patients suffering from malignant neoplasms. In a case of sarcoma of the forearm, a case of sarcoma of the thigh, and a case of sarcoma of the abdomen the percentage of eosinophiles ranged from 7 to 10 per cent.

Neusser considers eosinophilia as one of the diagnostic signs of sarcoma of the bone-marrow, and he finds that it is met with in osteomalacia.

In one case of lymphosarcoma of the neck, with metastasis in the bone-marrow, he found 60,000 eosinophiles per c.mm.

Exclusion of the splenic function may also be followed by eosinophilia. After the internal administration of camphor in two cases of

chlorosis Van Noorden noticed an increase of eosinophiles up to 9 per cent. In bronchial asthma Gollasch has found from 10 per cent. to 20 per cent. eosinophiles. In skin diseases (prurigo and psoriasis) Canon noticed in a large number of cases 17 per cent. eosinophiles. In a marked case of urticaria Lazarus found 60 per cent. eosinophiles.

Laptschinsky[20] mentions the presence of a very large number of coarsely granular leucocytes (eosinophiles?) in a case of relapsing fever.

Gabritschensky found 11 to 22 per cent. eosinophiles in a patient with emphysema, while Fisk reports 14.6 per cent. and Billings 53.6 per cent., the latter out of 8300 cells. Fuchs and Teichmüller found many eosinophile cells in various forms of bronchitis.

Zappert found 8 to 19 per cent. of eosinophiles in four cases of uncomplicated chorea. Cabot also refers to twelve cases showing eosinophilia, and in myxœdema the same observer found 5 per cent. and 4 per cent., respectively, in two cases.

Hock and Schlesinger found 20 per cent. in one subject suffering with rhachitis, and Weiss 16 per cent. in the same disease.

Loos claims that in syphilis the eosinophile cells are increased, especially when the eruption is extensive.

Klein has recently reported a case of hemorrhagic septicæmia in which the eosinophiles reached 76 per cent. in the pleural exudate and 40 per cent. in the blood. Aporti found as high as 18 per cent. eosinophiles during the intermittent pyrexia at the end of the disease.

Turk found 5 per cent. eosinophiles in a case of measles during the second week.

Ewing states that the eosinophiles reach a maximum of 8 to 15 per cent. in the second or third week in scarlet fever, decline slowly, and reach normal at about the sixth week.

To Professor Coplin I wish to extend my sincere thanks for the interest he has shown in the case and for the photomicrographs presented.

I also wish to extend my thanks to Drs. Butler and Dorsett, resident physicians of St. Joseph's Hospital, for their interest manifested in the case and assistance in perfecting the blood-counts.

Discussion by Dr. Coplin. At the Boston meeting of the Association of American Pathologists I suggested the possibility of a recrudescence of lesions. The evidence upon which to base such a view in this case is by no means conclusive, but may be briefly summed up as follows:

1. A more or less local, active lesion (trichina, œdema, myositis, etc.), with trichina present in other muscles, which at the time manifest no symptoms.

2. The absence of marked eosinophilia, so commonly observed in the initial outbreak of trichinosis.

3. The history of injury and the well-known fact that injury induces inflammation of fully encapsulated, but quiescent, parasites of other but similar types—*e. g.*, echinococci.

4. The evident pericapsular lymphoid accumulation, which is apparently most evident around the older capsules, thereby establishing to my mind that such active inflammatory phenomena as are present at this time are not of necessity related to the presence of living trichinæ.

The assumption that different broods of parasites are disseminated from a single mother may seem at first to negative the suggested view that recrudescence is possible, but it does not appear to me as conclusively inconsistent with the idea expressed. It seems quite difficult to understand exactly how one brood could infiltrate some muscles of one extremity to the exclusion of other muscles.

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HISTOLOGICAL DESCRIPTION OF AN EYEBALL, WITH DROPSICAL DEGENERATION OF THE NUCLEI AND PROTOPLASM OF THE ROD AND CONE VISUAL CELLS OF THE RETINA WHICH CLINICALLY SIMULATED GLIOMA.

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THE following case presents features, both clinical and pathological, of sufficient interest to render its record worthy:

A boy, aged two years, was brought to one of us (Dr. de Schweinitz) on January 20, 1900, by his physician, Dr. William L. Estes.

History. The boy is the youngest of six children, all the others being girls. He has always been puny, but has never suffered from any serious

illness; he has much trouble with his digestion. The cause of death of the child's paternal grandmother is not known except that it occurred from some acute disease; his maternal grandmother is said to have "died of exhaustion." The paternal grandfather died from a complication of liver and renal affections; the maternal grandfather, now over seventy-five years of age, is in excellent condition, suffering only from occasional attacks of gout. The child's father is an active business man; his mother is somewhat nervous, but in fair condition. She and one daughter suffer from chronic blepharitis.

About two weeks prior to the child's visit for ocular examination it was noted that the pupil of the right eye was slightly dilated, and that through it a yellowish-brown mass was visible. There was no history of injury and none of any affection, local or general, to which this change in the eye could be attributed.

Examination. The child was distinctly undersized and exceedingly pallid, but physical examination failed to reveal chronic disorder of any of the organs of the body, with the exception of the diseased eye, and the only constant menace were attacks of indigestion, which doubtless were frequently brought on by ill-considered diet.

Examination of the Right Eye. Vision was lacking, and light perception could not be demonstrated. The external appearance of the eye presented no anomalies. The pupil was semi-dilated and fixed, but the iris was of good color, resembling that of the opposite side; synechiæ and inflammatory deposits were wanting. The iris responded to a mydriatic, although the pupil did not dilate *ad maximum*. Well forward in the vitreous chamber and behind the pupil space could be seen a yellowish or rather yellowish-pink mass, over which some large vessels could be detected. This apparently filled the entire ophthalmoscopic field, and no red reflex could be demonstrated, nor was there any sign of undetached retina. The tension of the eye was normal or about equal to that of the eye upon the opposite side. The clinical appearances were those of glioma, and the opinion was expressed that the eye should be enucleated. The patient was also examined by Dr. Herman Knapp, who thought the disease was glioma, and, in any event, recommended the enucleation of the eyeball.

Examination of the Left Eye. This was normal in all respects.

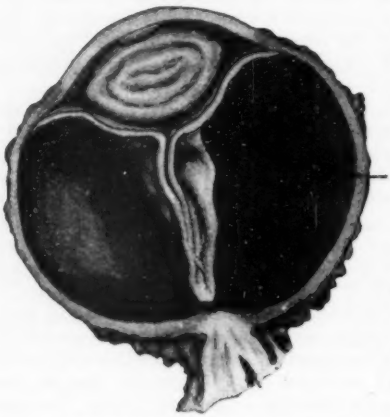
The eyeball was enucleated on January 27, 1900; chloroform narcosis. The healing was prompt, and there was absolutely no unfavorable sign at any period of the convalescence. After the original shock attending the operation of enucleation had passed away, and the necessity for frequent dressings of the eye had disappeared, the child seemed to gain in nutrition. This improvement was in a measure attributed to the removal of the eye. A recent letter, however, from Dr. Estes, the child's physician, states that "he has not improved in nutrition or growth markedly since the operation." He further states, however, that much of the child's indigestion, from which he still suffers, is due to injudicious feeding and indulgence in sweets. There has been no sign of recurrence *in loco* and no metastasis. Once, for a short time, a small œdematous swelling appeared at the bottom of the orbit. This, however, has entirely passed away, and was probably due to the pressure of the edge of the artificial eye. At the present writing, eighteen months after the operation, the child appears to be normal in all respects.

The enucleated eye was immediately placed in a solution of formalin, and after hardening divided and submitted to macroscopical and microscopical examination.

Pathological Report.

Macroscopical Examination. The eyeball measured 21 mm. in its antero-posterior and 22 mm. in its vertical and horizontal diameters. It was divided into two halves by a section passing in a horizontal plane through the centre of the cornea and the optic nerve. The retina was folded closely together in the centre of the globe, attached only at the optic nerve entrance and at the ora serrata. It was moderately thickened, but presented no distinct trace of tumor formation. (Fig. 1.) The subretinal space was filled with a grayish-white exudate; the choroid was in its normal position, and showed on its inner surface on the temporal side a narrow linear pigment mass. Aside from this, no cause could be seen for the retinal detachment. One-half of the specimen was mounted in glycerin jelly, the other half embedded in celloidin for microscopical section.

FIG. 1.



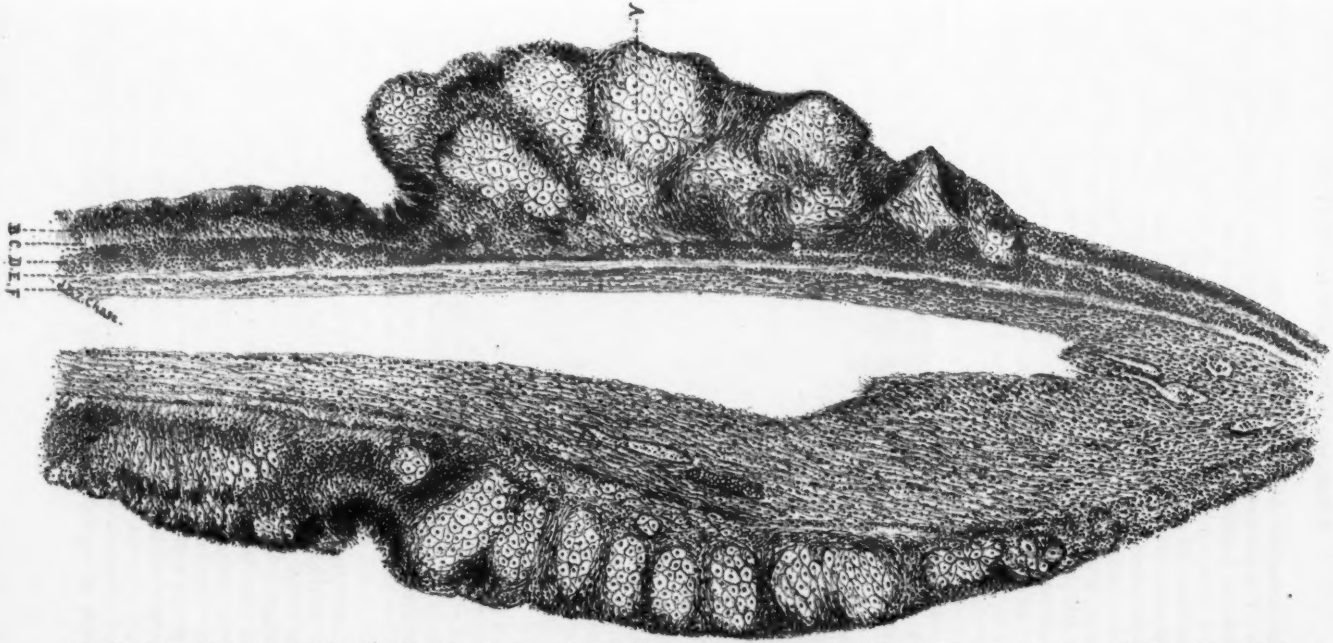
Detached and thickened retina.

Microscopical Examination. The cornea, anterior chamber, ciliary bodies, choroid, and sclera are entirely normal. The iris is partly adherent to the lens, but is otherwise normal and shows no sign of previous infiltration. The retina is in close apposition to the posterior surface of the lens, and the very small vitreous cavity contains a few fine, homogeneous bands, crossing between the inner surface of the retina, which include an occasional oval cell.

The subretinal space is filled with a homogeneous exudate which stains pink with the eosin. Embedded within it are a large number of enormously swollen cells, each of which possesses a shrunken, distorted nucleus, coarse, highly refractive granules, and masses of pigment. The pigment is in fine rods, and distinctly of the type peculiar to the retinal pigment cells. In many places all that can be recognized of the cell is a large, oval vacuole in the exudate, which contains an isolated nucleus and scattered pigment granules. These cells are especially numerous on the temporal side. They have remained attached to the outer edge of the exudate, and consequently have been separated from the choroid by the shrinking of the former, but they evidently represent the line of pigment described on the surface of the choroid in the macroscopical report. The retinal pigment cells form a regular layer, which has remained adherent to the surface of the choroid.

The optic nerve is partially atrophied, but the majority of nerve-fibres retain the Weigert nerve-sheath stain.

FIG. 2.

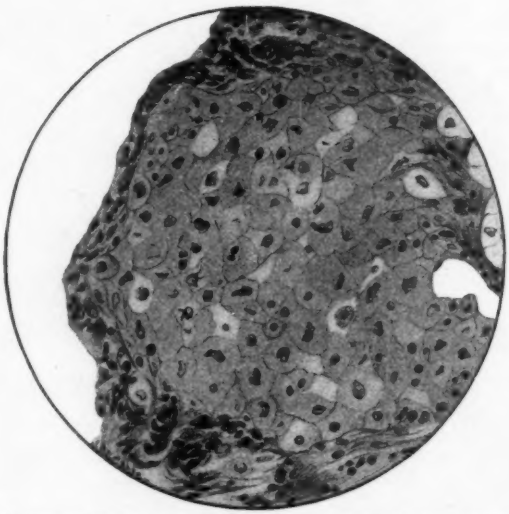


A. Cavities in the external nuclear layer of the retina. B. External nuclear layer. C. External granular layer. D. Internal nuclear layer. E. Internal granular layer. F. Ganglion and nerve-fibre layer. Leitz-Wetzlar objective 3, oc. 3.

The principal changes occur in the retina. It is moderately thickened. The nerve-fibre layer is œdematous, and the supporting fibres of Müller are prominent. The ganglion cells are in an advanced condition of chromatolysis; they are distorted and shrunken, their processes are broken abruptly off, the Nissl granules are absent, and the cell bodies show numerous vacuoles. The rods and cones have entirely disappeared, and the fibres of Müller extend in the form of fine arcades a short distance beyond the limitans externa, and in places show the presence of nuclei similar to those in the external nuclear layer. (Compare Deutschmann's[1] article, *Archiv f. Ophthalmologie*, xxv. 3, and Tartuferi's,[2] in *Centralblatt f. die med. Wissenschaften*, 1882, xx. 801.)

In the nasal half of the retina, commencing 1 mm. from the nerve entrance and running for a distance of about 1.5 mm., is a series of

FIG. 3.



Detail of structure from A, Fig. 2, showing large flat cells with degenerated nuclei. Leitz-Wetzlar objective 7, oc. 3.

cyst-like spaces, each measuring 0.16 to 0.25 mm. in diameter, situated in the position of the external nuclear layer, which is here absent. A similar series begins in the temporal leaf 3 mm. from the nerve entrance and extends for a distance of 4 mm. (Fig. 2.) For convenience, these spaces have been represented in the drawing as being exactly opposite. In these positions the retina is thickened to nearly double its normal width. The cavities are bounded laterally by the thickened fibres of Müller and externally by the limitans externa retinæ. They are crossed by a fine reticulum, which at first glance gives the appearance of a network of anastomosing fibres of supporting connective tissue. More careful examination, however, showed that this reticulum represents the walls of large, flat cells measuring between 15μ and 30μ in diameter,

each of which contains a distorted and degenerated nucleus either at the centre or near the periphery; the cell body has a finely granular appearance, more prominent in some cells than in others, but contains no large refractive granules. (Fig. 3.)

In sections stained by the Weigert fibrin-method the cell outlines do not retain the stain. The half of the eyeball which was mounted in the jelly for a macroscopical specimen was subsequently removed from the cup, and a portion of the retina was stained with osmic acid, but the cells were not blackened. The condition was traced through a series of sections, and the same changes were found in approximately the same position. The retina exhibits no sign of inflammatory process.

The explanation of these changes is difficult. The following possibilities have been suggested:

1. They represent a fine network of fibrin such as has been frequently described in retinitis albuminurica. The entire absence, however, of signs of inflammation in the retina, of round-cell infiltration about the vessels, of exudation or hemorrhage, and the negative reaction of the fine network to the Weigert stain, as well as the complete difference in the appearance of the cavities, render this theory untenable.

2. The condition is due to œdema of the retina, the spaces representing cavities between the separated fibres of Müller. Such cystic cavities have been very frequently described in the fibre layer of the retina, in detachment of this membrane, by Leber,[3] Müller,[4] Nordenson,[5] Poncet,[6] Caudron,[7] etc.; but in only two cases have there been found in the outer layers changes similar to those existing in our specimen. In 1882 Tartuferi[2] described, in a case of hemorrhagic glaucoma, "vacuole formation in the cones, due to œdema, which later may widen out and communicate through openings in the limitans externa with spaces in the external nuclear layer, which are likewise filled with serum and surround the cone nucleus on all sides, so that the latter is separated from the neighboring elements." In a case published by Webster,[8] in 1887, Van Gieson speaks of "numerous small, spheroidal cavities in the outer nuclear layer filled with fluid and containing delicate, flattened connective tissue fibres, with wing-like anastomosing processes. These are the fibres of Müller, exposed by the disappearance of the outer nuclei. These small cysts are enclosed between the limitans externa and the inner nuclear layer. There are similar cavities in the inner nuclear layer, but fewer in number." No plates accompany either paper. In our case, however, the minute spaces do not represent cavities formed by the separation of the fibres of Müller, but are distinct cellular elements. In this opinion we are supported by Professors Simon Flexner and George A. Piersol, of the University of Pennsylvania, who have studied the sections carefully. The cellular nature of the process is shown especially where the peculiar formation begins. A few isolated, swollen cells appear in the midst of the cells of the outer nuclear layer, and they gradually become more numerous until they fill out the large spaces described above.

3. They are fat, granular cells, such as are found in the degenerative lesions of the central nervous system (in softening of the brain, etc.). They contain, however, no refractive granules, and are not stained by osmic acid.

4. They are inclosures in an obliquely cut and folded retina of the cells described in the subretinal fluid. Now, although they are of

much the same size, they present an entirely different appearance. They contain no highly refractive granules, and only rarely any pigment, although the two forms of cells are separated in some places only by the limitans externa retinæ.

5. They are due to the burrowing of these cells into the retina, where they have undergone secondary proliferation and lost their pigment and refractive granules. Such a process, however, would be hard to understand.

6. They are a proliferation of the nuclei of the external nuclear layer, and represent a true tumor formation. Such a condition is especially suggested by the overlapping of portions of the retina, as though from an active cell proliferation. The change, however, has occurred in two remote portions of the retina at the same time, and the cells, with their degenerated nuclei, do not have the appearance of actual proliferating cells. A tumor of this character would be unique.

7. They are due to an œdematous or dropsical degeneration of the nuclei of the external nuclear layer. The latter are the nuclei of the rod and cone visual cells, modified epithelial cells derived from the embryonic epiblast, and would be subject to the changes observed in such cells. That the condition is not a fatty metamorphosis is shown by the absence of granules and the negative reaction to osmic acid. Myxomatous degeneration may be likewise excluded, as the cells do not stain typically with hæmatoxylin or thionin. Œdema of the retina is one of the most frequently described conditions in detachment, and in this respect our case forms no exception to the rule. Therefore, it is possible that we have here a localized dropsical degeneration of the nuclei and surrounding protoplasm of the visual cells. This we believe to be the most probable explanation of a change which is certainly of great rarity, if it is not unique. At the same time we cannot exclude entirely the possibility that it is a true tumor formation, and must leave the matter for time and future observation to decide.

A word may be said in regard to the diagnosis from the clinical stand-point. As has been particularly pointed out by Mr. E. Treacher Collins, the following conditions may be mistaken for glioma: Persistence of the posterior part of the fœtal fibrovascular sheath of the lens, masses of tubercle in the choroid, and inflammatory or purulent effusion into the vitreous, following retinitis or cyclitis, usually with detachment of the retina. Although well-marked differential diagnostic points have been established between these affections and glioma of the retina, it is frequently very difficult to come to a definite decision, and, therefore, it is an excellent surgical rule that the eye should be enucleated in case of doubt.

In June, 1889, De Wecker[3] called the attention of the Society of Ophthalmology of Paris to a danger to which young subjects attacked with white degeneration of the retina, or retinitis circinata, are exposed, inasmuch as the disease may be mistaken for glioma. He believes that this affection is not necessarily confined to the eyes of adults, but may be seen also in infants. Among three observations he cited the case of a girl, aged six years, under his care for two

years, during which time, although the lesions resembled glioma of the retina, they had not increased, and the intra-ocular tension had not been augmented. This eye was afterward enucleated by one of De Wecker's confrères, who, in addition to the plaques of atrophied choroid and degenerated retina described by De Wecker, found a small ossifying fibroma. In the present instance the clinical appearances were exactly those of glioma; and, although, fortunately for the child, this malignant form of growth was not discovered with the microscope, in the light of the report already cited, it is not absolutely sure that tumor formation is entirely absent. The enucleation, therefore, was a wise procedure.

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COMBINED CLINICAL REPORT OF TWO CASES OF AORTIC ANEURISM TREATED BY MEANS OF SILVER WIRE AND ELECTRICITY.[1]

(1) THE TREATMENT OF AORTIC ANEURISM BY MEANS OF SILVER WIRE AND ELECTRICITY, WITH REPORT OF CASES.

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THE literature of this subject has been so thoroughly collected and studied by D. D. Stewart and Guy L. Hunner in their recent elaborate papers[2] that it is unnecessary for me to go over the ground again. I shall merely mention a few prominent points.

The method of treating aneurisms of the aorta by means of a fine

[1] Read before the American Surgical Association, at Baltimore.

[2] Hunner, Johns Hopkins Hospital Bulletin, November, 1900. Stewart, AMERICAN JOURNAL OF THE MEDICAL SCIENCES, October, 1892; Ibid., August, 1896; Philadelphia Medical Journal, November 12, 1898.

wire introduced through a hollow needle and coiled within the sac was first suggested and carried out in 1864 by Charles H. Moore, of the Middlesex Hospital. Corradi, in 1879, added much to the effectiveness of the procedure by passing a strong galvanic current through the wire, thus promoting more rapid and firm coagulation. It has hence been designated "the Moore-Corradi operation." D. D. Stewart, of Philadelphia, has done more than anyone else to bring the operation into prominence, perfect its technique, and emphasize its possibilities.

There have been reported, according to Hunner, fourteen cases in which wire alone was used, with three recoveries, although one patient subsequently died from rupture of the aorta below the tumor, and twenty-three cases in which both wire and galvanism were employed, with four recoveries; in all, thirty-seven cases, with seven recoveries. There have also been a number of encouraging improvements.

Apparently this is not a brilliant showing, but it must be borne in mind that many of the aneurisms were extremely unfavorable for operation. In some instances the sac was fusiform or had several compartments, or was on the point of bursting; while in others the patients were nearly dead from exhaustion or from pressure upon the trachea, and occasionally a poor technique seems to have been directly at fault. Failure, under such circumstances, cannot excite surprise; and it must not be lost sight of that no other treatment can furnish results at all comparable to these.

To obtain the best results it seems evident that the cases should be carefully selected. The aneurism should be sacculated, not fusiform; without too large an opening, and with but one compartment. It should not have progressed too far toward rupture, and the patient should have sufficient vitality to enable him to live through the tedious process of cure. It is scarcely necessary to state that the operation is practically confined to aneurisms not amenable to ordinary surgical procedures—*i. e.*, aneurisms of the abdominal aorta, the thoracic aorta, the innominate artery, etc., the first named requiring a preliminary cœliotomy.

An accurate diagnosis is of the utmost importance; but, in spite of the greatest care, mistakes are apt to occur. We will hope, however, that when the operation comes into more general use better methods of investigation will arise. Perhaps the X-ray may be of service, as insisted upon by Dudley Tait, of San Francisco.

In addition to reporting two cases of my own, my object is to discuss several points pertaining to the technique, which, according to Stewart, is briefly as follows: Asepsis, local anæsthesia, insertion into the aneurism of an insulated hollow needle, through which is introduced silver wire (about No. 27). It must be well drawn and wound on a spool, to increase its tendency to coil, and from 3 to 20 feet in length, according

to the size of the cavity. The wire is then attached to the positive pole of a galvanic battery, the negative electrode, large and well moistened, being applied to the back, and a current of 70 to 80 milliampères passed for from half an hour to an hour and a half. The wire is then cut close to the skin and sunk beneath the surface.

Let us consider in detail some of the questions involved.

Character of the Wire. Most operators agree that silver wire is preferable to any other. It is easily obtained, inexpensive, is a good transmitter, and becomes sufficiently corroded by the passage of the electric current to insure adhesion of the clot. Steel is said to be too brittle, and soft iron too easily disintegrated. The wire should be fine (about No. 27), because fine wire coils more readily and offers less resistance to electricity and to contraction of the sac. Silver wire varies much in elasticity according to the amount of copper which it contains and the degree to which it is drawn. Hunner lays great stress upon the use of highly drawn wire (from Nos. 8 to 27), containing seventy-five parts of copper to the thousand, making it so hard and springy that it readily retains the coil imparted to it. Stewart also insists upon the use of wire which has been drawn, and both he and Hunner strongly advise winding the wire upon a spool before inserting it. These ideas are founded upon the manner in which various kinds of wire deport themselves when introduced into glass flasks. They claim that much can be learned in this way regarding the behavior of wire in an aneurism, and that no one should operate upon the living until numerous experiments of this kind have been made.

Hunner, in his beautifully illustrated article, shows how ordinary undrawn silver wire arranges itself in large circles around the periphery of a flask without filling the centre, while his highly drawn elastic wire coils itself in the middle of the receptacle.

It is somewhat questionable, however, whether it is advisable to have most of the wire in the centre and comparatively little in contact with the walls of an aneurism. The firmness of a clot, its stability, and its capacity for organization must depend somewhat upon the support derived from mural implantation. A clot suspended in the centre of the sac would be of little use, however satisfactory its consistency. In addition we must not forget that an aneurism cures itself by the deposition of clots about its circumference, and that the electrolytic effect of galvanism upon the sac, as demonstrated by Macewen, promotes the accumulation of leucocytes and the formation of firm, white, readily organizing coagula.

But aside from these more or less theoretical considerations, it occurred to me that glass flasks do not represent actual conditions, and that we can draw no reliable conclusions from their employment. In repeating Hunner's experiments—which I did with wire obtained directly from

him as well as with ordinary silver wire, both drawn and undrawn—I found that the strands slipped easily about within the smooth, hard, glass-lined cavity, and arranged themselves always in a definite manner, as he has described. The inside of an aneurism, however, is not hard and smooth; it is often irregular and coated with more or less rough layers of clot, so that the wire would easily catch and be deflected from its natural course; also, the coils could not rotate within the cavity and adjust themselves when pushed upon by the entrance of a fresh portion, as occurs in a flask.

In order to represent more accurately the conditions as they really exist, I obtained a large Colorado turnip, the size and shape of a flask of 500 c.c. This I hollowed out through an opening large enough to observe what took place within. The walls were of the consistency of an old, firm clot, and of about the right irregularity and roughness. After running a hollow needle through the side of this "aneurism" I inserted various kinds of wire. My observations convinced me that the glass-flask method had led to erroneous and misleading conclusions. Ordinary undrawn silver wire under these new conditions coiled itself backward and forward and around through the cavity, not only covering the walls to a satisfactory extent, but filling the centre also—totally different from its conduct in a flask. Even wire not previously coiled did almost, if not quite, as well in this regard. The stiff, springy wire obtained from Dr. Hunner formed a good network within the turnip, also, but not so good as the ordinary wire. The meshes were not so evenly distributed over the walls and centre, and when the wire had been wound upon a large spool it was still less effective, quite large open spaces being left within the curl-like rings.

When the needle was directed away from the opening there was little or no tendency on the part of the ordinary wire to escape through it; but a coil or an end of the Hunner wire would often spring suddenly through the hole, like a Jack-in-a-box. I feel that a real danger lurks in these sudden movements from dislodgement of clots, penetration of the aorta, etc. The phenomenon was, of course, still more marked when the needle was directed toward the opening. Through a trap-door in the turnip I was able to extract the coils without disarranging them. I observed that a coil of ordinary soft wire could easily be squeezed together in the hand, and that it remained as it was moulded, while a coil of drawn and alloyed wire (Hunner) was so springy that it resumed its original shape as soon as the pressure was removed.

These experiments convinced me that Hunner and others are mistaken in their deductions. Instead of hard, elastic wire being superior to the ordinary soft variety, the advantage lies just the other way. The elastic wire really possesses certain disadvantages: It kinks in a more annoying manner and breaks more readily; it does not coil so satis-

factorily within an aneurism; it is more liable to protrude, sometimes with force, through the aortic opening; its sudden movements during introduction may dislodge dangerous clots; and its inherent springiness would tend to prevent contraction of the sac wall, although I do not lay so much stress upon this as do some. Soft, unalloyed silver wire has another advantage over the harder variety in that it more rapidly loses in size under the influence of the electric current, and becomes much rougher and more brittle. I found this difference quite marked in a séance of but twenty minutes. Wire thus acted upon breaks and crumbles to pieces readily and can offer no appreciable resistance to contraction.

In the course of my experiments I noticed another point of some importance. If the canula is inserted too far into the sac, as we are apt to insert it, the wire has a strong tendency to curl up against the further wall, leaving a more or less clear space surrounding the place of entrance—a spot which often needs strengthening more than others. This may be avoided by just penetrating the aneurism with the needle, and no more.

Quantity of Wire to be Used. Stewart, Hunner, and others confidently assume that it is far better to employ a small amount of wire (from 3 to 10 feet, or at most 20 feet) than a large amount. They are so sure of this that they attribute some of the failures which have occurred to a disregard of this principle. Their theory is based upon the following considerations: 1. Large amounts of wire are unnecessary, smaller pieces producing clots which are just as satisfactory. 2. Much wire tends to prevent contraction of the sac wall, upon which depends the cure. 3. The reported recoveries were obtained with small amounts of wire.

At first sight these points appear convincing, but upon closer inspection it develops that both reasoning and conclusion are open to question.

It seems sufficiently evident that the greater the quantity of wire used the more certainly and quickly will a clot form, and the more firm and lasting will it be, lodged as it is among the meshes of the wire. In fact, if every portion of the sac could be filled completely with wire there would be no room for blood at all, and the aneurism, as such, would cease to exist.

Aneurisms are definitely cured by the gradual replacement of coagula by fibrous tissue, which slowly but firmly contracts. The contractile power of such a mass is certainly considerable, as is seen in scar-tissue in other portions of the body, and is not to be seriously impeded by the presence of a more or less loose coil of fine silver wire, especially if the wire be soft and devoid of spring. This can be more clearly appreciated after crushing such a coil in the hand. Hunner and Stewart admit the power of this contractile force when they assert that it is

dangerous to use steel wire because of the likelihood of its breaking and penetrating the sac wall. If steel wire can be broken, soft silver wire can certainly be easily bent into a small compass. But even admitting that contraction would be to a certain extent impeded, it is hard to see what actual harm would be done in the presence of a firm, well-organized clot. To be sure, contraction would sooner reach its limit, a larger bunch of tissue remaining; but as long as this consisted of closely coiled wire, connective tissue, and fibrin no objection could be made to its mere presence. It can here be clearly appreciated that soft, undrawn wire would possess an advantage over that which is hard and springy, as the latter would cause a continuous centrifugal pressure.

It is true that the reported cures have been obtained in cases where small amounts of wire were used; but this fact is misleading, as is shown by a glance at the records as given by Hunner himself. According to these, wiring has been resorted to 37 times, much wire being employed in but 7 instances—*i. e.*, 78 feet, 75 feet (in two sittings), 32 feet, 33 feet, 225 feet, 150 feet, and 32 feet (in three sittings). It is, however, questionable whether such lengths as 32 feet and 33 feet should be considered as "much" wire, when we remember that both Stewart and Hunner recommended as high as 20 feet under certain circumstances. The figures would then stand 33 to 4. For the sake of argument, however, we shall assume that much wire has been used in 7 cases and a small amount in 30. But 4 of these 7 cases must be excluded because of features rendering their consideration absolutely unfair. They are as follows:

Moore's case (78 feet); died of sepsis.

Cayley's case (75 feet); *two* sacs, with a *very* large opening into the main vessel.

White and Gould's case (32 feet); *two* sacs; fusiform aneurism; death from sloughing due to pressure from without.

Abbe's case (150 feet); reversed current in middle of sitting and probably softened clot already formed.

This leaves but three cases—one in which 33 feet were used, one 32 feet, and one 225 feet—*i. e.:*

Hulke's case (33 feet); "desperate condition;" wire did not fill entire sac, which continued to expand in empty portion. If this case shows anything, it is that not enough wire was used.

Finney's case (32 feet in three sittings); no autopsy, and hence we know nothing of the real condition of things within.

Roosevelt's case (225 feet); improved, the tumor becoming firmer; died on twenty-second day, probably of embolism.

It is thus seen that a really large amount of wire has been used in but one case which can be called at all favorable, and in this the patient lived twenty-two days, finally dying of what appeared to be

embolism—an accidental complication. In the meantime the patient had improved and the tumor grown more firm.

Hence we are forced to conclude that the employment of considerable wire, within reason, has some theoretical advantages without counterbalancing disadvantages, and that it has never been given a fair trial, the question of "much wire or little wire" still remaining an open one.

Strength of Current. Stewart and others insist upon the use of rather a strong current—about 70 to 80 milliampères, as a rule. Hunner objects to this, regarding it as dangerous from disintegration of the sac wall, or even of the aorta itself in case of misplacement of the wire. Hunner claims to have demonstrated by his experiments that a current of 10 milliampères, continued for fifteen or twenty minutes, produces practically as large and effective a clot as one of 100 milliampères used for a much longer period. This seems hardly probable on the face of it, and, in addition, in one of my operations coagulation did not occur, as evidenced by the movements of the needle, for some thirty minutes after the beginning of the operation. Hunner's experiments were made in rapidly moving blood in a dog's aorta. Under such conditions we know that clotting of blood is difficult to obtain, as shown by several autopsies in which wire accidentally introduced into the aorta remained perfectly clean; and it must also be considered that, where the circumference of the clots obtained is so small, relative differences are hard to estimate which in larger clots would be quite marked. Such an experiment is hardly a fair test.

In my opinion, more accurate data can be obtained by employing coagulated blood, because in aneurisms the blood does not move with anywhere near the rapidity that it does within the aorta; and, in addition, it has been noted in many operations that blood often clots shortly after turning on the electric current; hence we often operate not only in comparatively stagnant blood, but even in a coagulum.

It will also be admitted, I am sure, that the solid clot, which is, as it were, burned onto the wire, counts for much more in the ultimate result than the soft coagulum which may form around it. I found by conducting experiments in clotted blood that the diameter of the area acted on by a current of 100 milliampères was several times greater than that effected by a current of 10 milliampères during the same length of time—twenty minutes.

The danger of injuring the aorta or the sac wall by a current of 70 or 80 milliampères is probably very remote (so far as known, it has never occurred), and is more than counterbalanced by the advantages obtained.

(As a mere matter of interest, in blood prevented from coagulating by the addition of sulphate of magnesia, I was able to produce but a small amount of coagulum, closely adherent to the wire, and I could

detect little, if any, difference between the effects of currents of high power and those of low power.)

According to both Stewart and Hunner, the chief danger in operating upon aneurisms with wire is the clotting of the blood in but a portion of the sac, thus exposing the remainder to increased pressure and possible rupture. This hypothesis, however, does not seem reasonable. A sacculated aneurism, according to the size of its opening, contains a more or less comparatively quiescent pool of blood. When this pool is struck by an impulse from the heart as it passes through the aorta it expands, and this expansion, so say the laws of hydrostatics, is equal in all directions. If a clot forms in a portion of the cavity, this does not increase the pressure upon the remainder of the wall. It does not even increase the general blood-pressure, because nothing is added to the total quantity of blood. In fact, the general blood-pressure, together with that in the aneurism, is often lowered by the considerable loss of blood which may take place during the operation.

In order to convince myself thoroughly on this point I fastened a rubber glove to a fountain-syringe filled with water and elevated the syringe. The glove represented an aneurism, and the force of the water in the douche bag took the place of the pressure of the blood, this force being measurable by the height to which a fine stream of water was projected through a hole in one of the glove fingers. After the insertion of a large, solid body within the glove, to represent a clot, it was noted that the stream projected to exactly the same height as before, thus demonstrating that there was absolutely no increase of pressure.

Conclusions. 1. Considering the inefficiency of medical treatment, and the comparative efficiency of the use of silver wire and electricity, it is probably better to proceed to the latter at once without wasting valuable time upon the former. This seems all the more desirable when we consider that wiring is not a very dangerous process, and that it is in the early stages of an aneurism, when the sac is still firm and the patient is in good condition, that the best prospect of cure exists.

2. Soft, undrawn, unalloyed silver wire devoid of spring—wire just as it comes from the shop—is preferable to the hard, highly drawn wire alloyed with copper and full of spring. It is hardly necessary to previously coil the wire.

3. It is still an open question as to which is preferable, a large amount of wire or a small amount, with the theoretical advantages in favor of the former.

4. A strong electric current is apparently preferable to a weak one.

5. The canula through which the wire is introduced should be inserted just within the sac, and no further.

6. There is little if any danger of bursting the aneurism from increase of pressure due to coagulation in a portion of the sac only.

The report of the two cases in which I have utilized this method will be found in full in the accompanying article by Dr. J. N. Hall.

To Dr. E. P. Hershey, of Denver, the first to successfully wire an aneurism in Colorado, I desire to express my indebtedness for valuable assistance.

(2) REPORT OF TWO CASES OF ANEURISM TREATED BY WIRING AND ELECTROLYSIS.[1]

By J. N. Hall, M.D.,
of Denver, Colo.

The treatment of aortic aneurisms in general is extremely unsatisfactory, and, from the very nature of the pathological process underlying them, must always remain so to a greater or less degree. Always originating in localized degeneration of the arterial coats, this degeneration is but a few steps more advanced at the seat of the aneurism than elsewhere, for in the internal arteries traumatic injury rarely, if ever, plays the part which it exercises occasionally in peripheral vessels. We should apprehend distinctly that cure of aortic aneurism does not signify as much as cure of many other conditions does, since we cannot hope to cure the general arterial deterioration; yet many patients the subjects of this disease live in comparative comfort for a considerable time, and we may hope, in favorable cases, to prolong this time materially.

Surgical measures are necessarily limited to those aneurisms accessible from the surface, either directly or after opening the abdominal wall. I speak to-day chiefly of those springing from the anterior or lateral surfaces of the ascending portion of the arch of the aorta and of such sacculated character that clotting of their contents would not shut off the direct current through the aorta. A very small percentage of the aneurisms of the arch are of this nature, but, where the diagnosis can be made, the gloomy prognosis of medical treatment should lead us to offer surgical relief.

A review of the characteristics of the aneurisms amenable to such operative measures is of interest. It should be noted that aneurism of the innominate artery must be considered in this connection, since it is intimately associated in many instances with that of the portion of the aorta mentioned.

In a general way, the aneurisms of the ascending portion of the arch eligible for surgical treatment must be those presenting chiefly the signs of growth anteriorly or laterally. These signs are entirely consistent

[1] Read before the American Therapeutic Society at Washington.

with sacculation of the aneurism. Even though sacculated, if the tumor project from the posterior wall of the ascending aorta it must be looked upon as ineligible for such treatment, not only because of its inaccessibility, but because of the difficulties in the way of the diagnosis.

The signs and symptoms found in the aneurisms mentioned as being suitable for treatment are in general those that indicate growth toward the surface, with involvement of the parietes, rather than toward the centre of the chest, with pressure upon internal organs. Thus we have pressure upon the ribs or sternum, with consequent neuritic pain, bulging of the bony parts, a pulsatile and expansile tumor, dulness, diastolic shock, and perhaps auscultatory signs or the inflammation of the skin which precedes ulceration, as in one of our cases.

It is not common in these cases to have dysphagia or pressure upon the trachea or bronchi. Hoarseness or aphonia from pressure on the recurrent laryngeal nerve, pupillary signs from involvement of the sympathetic, and tracheal tugging and diastolic shock, however, may all be present, as in the first case to be reported, without contraindicating the attempt or preventing a successful issue. Inequality of pulse in the two radials, with asynchronism, may occur, owing either to pressure upon or distortion of the orifice of the innominate or subclavian, or to the delay of the pulse-wave in the large sac.

It is quite possible that we may, in selected cases, disregard all of the signs of intrathoracic pressure, for they may come from the pushing back of the vessel by an aneurism bulging anteriorly, as was probably the case in the instance just mentioned, where even slight dysphagia existed.

I do not believe it advisable to attempt surgical measures in these cases unless other features of the case be favorable. Thus I advised against operation, some three years ago, in the case of a woman, aged fifty-six years. She had about such an aneurism as that in the case to be first reported, but a well-marked aortic regurgitation was present, with extensive atheroma. She had advanced tabes dorsalis also. The aneurism itself was eminently fitted for operation, but it was wholly unjustifiable in her condition. I think it possible that a slight aortic regurgitation may be due to deformity of the orifice or imperfect apposition of the leaflets caused by the aneurism, without actual valvular change, in which case treatment might be of especial value. It would be practically impossible to determine this fact, however, in most cases.

This case I report by courtesy of Drs. G. E. Tyler and Leonard Freeman, with whom I saw it at the Arapahoe County Hospital.

Case I.—D , miner, thirty-six years of age, was admitted August 16, 1900. He had syphilis at eighteen, later an attack of typhoid, with pneumonia, and, eight years ago, acute articular rheumatism of several weeks' duration. He had done hard manual labor all his life.

For seven months he had had pain in the right side of the neck. Four months ago a tumor appeared, and one month later his voice failed. There has been of late slight difficulty in swallowing. Pain is at times severe in the suboccipital and mid-scapular regions.

He was a well-developed laboring man, with healthy general appearance. A pulsating tumor existed in the base of the neck upon the right side, easily visible across the ward. It rose behind, and dislocated the right sternoclavicular joint, and was of the size and shape of a goose-egg.

The dulness around the base extended to the second interspace and the left edge of the sternum and slightly beyond the right edge of this bone.

Tracheal tugging and diastolic shock were present, although no diastolic shock was perceptible over the tumor. The right pupil was smaller than the left, and the right vocal cord was paralyzed. The pulse was from 84 to 100, temperature normal, urine negative.

After three weeks' rest in bed, on September 6th, 250 c.c. of 1 per cent. gelatin salt solution were injected, and on the 8th the same amount of 2 per cent. solution. Much after-pain resulted, and this treatment was abandoned. On September 17th, no improvement having been noted, and the patient rapidly getting worse, orthopnœa having developed, Dr. Freeman tied the right carotid and subclavian arteries under local anæsthesia by Schleich's solution, without material pain or shock, immediate union resulting. On September 21st, 23d, and 24th the 2 per cent. gelatin was injected as before.

On September 28th, no material improvement having resulted, the patient being worse off than before in many respects, and the skin over the aneurism being much reddened, 5 or 6 feet of No. 27 silver wire were introduced through a trocar by Dr. Freeman; it was the ordinary undrawn, coiled silver wire kept in the surgical supply houses. A current of 75 milliampères was applied for thirty minutes. The gelatin solution was injected on September 29th, 30th, October 2d and 3d, as before. The needle being imperfectly insulated, the skin was burned over an area the size of a silver dime. The needle puncture did not promptly close, and oozing of serum took place for a number of days. A tumor the size of a large olive, filled with serum, projected from the general contour of the aneurism. There was also a soft spot in the aneurism which persisted for many weeks. This was presumably also filled with serum.

The patient remained in the hospital for over four months, most of the time in bed, gradually improving, although the improvement was scarcely noticeable until the disappearance of the serous tumor. The tumor has steadily diminished until to-day (April 10, 1901) none is to be found. A slight pulsation has returned, through collateral circulation, in the right radial artery. The right hand is slightly cyanotic. The right pupil is moderately contracted. The voice remains husky, but is greatly improved. No dysphagia remains. Auscultation reveals nothing abnormal in the chest, while percussion still shows a slightly increased dulness over the region just below the right sternoclavicular articulation. The patient's collar pressed upon the skin over a prominent coil of wire and caused a small ulceration, with a fistulous opening leading down to the wire. This causes no trouble at present. The patient took moderate doses of potassium iodide for a long time.

It is of the utmost interest to me, in view of my recent study of the tracheal diastolic shock, to note that this phenomenon disappeared some weeks before the tracheal tug, during the retrogression of the aneurism.

I have elsewhere stated that I believe this sign—the shock of closure of the aortic valves transmitted through a fluid tumor to the trachea and manifested by a diastolic shock felt at the cricoid cartilage immediately after the tracheal tug—to be of value in distinguishing aneurism from tumor, although several instances have been reported of tracheal tug in connection with mediastinal tumor. The disappearance of the sign as the aneurism solidifies, with the subsequent disappearance of the tracheal tugging, was thus very gratifying to me as tending to confirm my position. The patient is at work as a day laborer, handling the pick and shovel, and finds no difficulty in this work. A skiagram fails to reveal the wire, since it is hidden by the shadow of the aneurismal contents.

In the early part of July, 1901, this patient returned with a portion of wire presenting at the mouth of the sinus. There was redness, swelling, and suppuration. I easily removed the wire, which was compressed into a small bunch. In a few hours a severe hemorrhage (in the nature of a secondary hemorrhage from ulceration?) took place.

This was followed at intervals of a few days by others. The aneurism rapidly re-formed, causing compression of the trachea, with coughing of blood and very difficult respiration. Although the man was in a desperate condition, another wiring was done on July 13, 1901. About fifteen feet of soft, uncoiled wire (No. 27) were used, a current varying from 50 to 70 milliampères being turned on for thirty-five minutes. The operation was well borne, but the dyspnœa became so extreme that death resulted in sixty-two hours. Autopsy revealed a firm, satisfactory clot filling the entire aneurism, with the wire passing in and out throughout its substance in all directions. The fatal result was due to pressure on the trachea, which had not been relieved by the operation.

Case II.—L., aged fifty-six years, hotel-keeper, was seen December 13, 1900, with Drs. Leonard Freeman and J. P. Roberts. Although he had been very active in athletic ways, his work had not been severe. He had not drank heavily, and there was no specific history. He was well developed and well preserved.

For six months he had pain in the upper chest, upon the right, shooting into the right side and arm. It was worse after exercise. Rest, and at times change of position, relieved the pain somewhat. During the past few days he has noticed a pulsating tumor over the aortic area, which led him to seek advice.

His voice and vision were normal. Upon examination we found a bulging tumor at the second right interspace, about three inches

across, the skin inflamed and angry in appearance, with marked visible and palpable pulsation. This was decidedly expansile in character. The ribs were not eroded entirely through, although the tumor crowded them forward. The heart-sounds here, as elsewhere, were normal, with the exception that the aortic second sound was sharply accentuated. The heart was normal in area and position. Pulse 108, respiration normal. The pupils were equal and responded to light. The larynx showed nothing abnormal. The urine was negative. A slight tracheal tug could be detected, but no diastolic tracheal shock. The lungs were entirely negative, excepting for the dulness over the seat of the aneurism. The hand was congested and slightly cyanotic.

The patient was sent to St. Joseph's Hospital, and, with rest in bed for ten days and small doses of potassium bromide, improved notably. The redness over the tumor subsided, and the prominence and pulsation were less marked. The pain diminished in corresponding manner.

On December 24th, Dr. Freeman introduced, under local anæsthesia by Schleich's solution, 22 feet of No. 27 silver wire of the kind mentioned, through a deeply introduced trocar, slightly to the outside of the centre of the protuberance caused by the aneurism. The blood spurted in a fine stream several feet high for an instant, but only a few ounces were lost as the wire was inserted. A current of 70 milliampères was applied for sixty-five minutes. Upon the first application the pulse slowed from 90 to 52, and the patient felt very faint, but he recovered immediately upon stopping it, and by raising it gradually to this point no further trouble ensued. The vagus had probably been too much stimulated. A hypodermic injection of strychnine was given at the time.

The bleeding ceased upon the application of the current. No effort was made to check the slight loss of blood at the beginning, as it was thought that clotting would be promoted by the lowering of the blood-pressure.

At the close of the operation the pulsation was notably less than at the beginning. No change in the auscultatory signs could be detected. Following the operation, rest in bed, subcutaneous injections of 2 per cent. gelatin solution, and small doses of potassium iodide were employed. Following the second operation the gelatin was given by enema daily. No very apparent results could be noted from these measures, however.

The tumor was decidedly reduced in size during the few weeks following the operation, and the patient's pain entirely disappeared. The expansile pulsation was no longer noted, although the aneurismal mass was lifted upward by the underlying aorta.

By February 20th it was evident that pulsation was increasing at the right edge of the tumor. Slight dilatation of the right pupil was noted, and an exceedingly soft systolic murmur was heard over the aneurismal area. Dr. Freeman therefore introduced, in the same manner as before, 8 feet of wire, and the current was applied for seventy-seven minutes at 70 milliampères and for fifteen minutes at 50 milliampères, with a short interruption because of irregular working of the battery.

The patient stood the operation well, and at its close the pulsation of the aneurism had practically ceased excepting at the extreme right.

By March 13th it was evident that some pulsation was to be found, but the canula withdrew only serum. Three feet of wire were introduced with difficulty, and the current at 75 milliampères applied for seventy minutes. On April 29th the tumor still pulsated at the right

border, and a deeply introduced canula brought blood at a depth of one and a half inches. No pulsation was to be noted in the flow, however, thus indicating that the blood existed in a quiescent pool in the centre of a thick coagulum. On the next day Dr. Freeman inserted 6 feet of wire without the difficulty which had led him to desist in the last attempt, and applied the battery at 75 milliampères for seventy-two minutes. After the first half-hour the pulsation communicated to the needle ceased. The patient was entirely comfortable after removal to his room.

Auscultation on May 1st showed the respiratory murmur to be normal throughout the chest. The soft systolic murmur persists over the tumor, with the moderate aortic accentuation. Heart otherwise negative. Radial pulses equal and synchronous. The tumor still lifts slightly with each heart-beat, but does not expand.[1]

The last-quoted case, although not yet complete, demonstrates conclusively to me that we should follow up the treatment as may be necessary. If an aneurism be of large size, only a portion of it may be solidified at the first attempt, and bulging may occur elsewhere, as in this case. This should be met by renewed introduction of the wire and electrolysis. I venture the assertion that more aneurisms will be cured in the future by repeated attempts than at the first trial.

I have not seen sufficient benefit from the injection of gelatin solution under the skin or per rectum to lead me to place confidence in it; still, being a safe measure, with some possibility of good, I think it may be properly used. If there were any way of becoming assured that the sac were cut off by fibrin or otherwise from communication with the aorta, I would introduce the solution into the aneurism. It is probably too hazardous a procedure as our knowledge of such matters now stands.

I do not think we have sufficient evidence upon which to base conclusions as to the size of the opening from the aorta, the chances of obtaining complete solidification, or the danger in operating. Patients really incur a considerable risk in any operative procedure, but an entirely justifiable one in view of the bad prognosis of the disease under medical treatment.

Although certain reporters have stated that great aid was obtained by means of the fluoroscope and radiograph in deciding as to the size, shape, and location of the aneurism, so little was gained in repeated attempts with our first patient that none was made with the second.

So much uncertainty must necessarily always exist regarding the shape, size, and parietal condition of an aneurism that uniformly good results must be impossible. The favorable outcome in some cases, however, is sufficient to encourage us to continue along this line.

[1] October 16, 1901. This patient continues in good health, and manages his hotel as formerly.

A CASE OF SYPHILITIC INSANITY AND ONE OF PARALYTIC DEMENTIA SIMULATING SYPHILITIC INSANITY;

WITH POST-MORTEM FINDINGS AND REMARKS ON THE DIFFERENTIAL DIAGNOSIS OF THE TWO DISEASES.[1]

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AND

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According to Fournier, about 21 per cent. of cases with tertiary syphilitic diseases show brain symptoms of syphilitic origin. Hielman states that of those infected with lues, from 1½ to 2½ per cent. get brain syphilis; he does not include general paresis and tabes in this estimate. Of these perhaps a quarter recover, another fourth improve decidedly; at least half die or only survive with grave disease.[2]

Not all cases of cerebral syphilis present mental symptoms; neither do all cases of insanity presenting a distinct history of syphilitic infection show characteristic mental symptoms or the somatic signs which are common manifestations of the syphilitic toxin. Syphilis may be an etiological factor in the production of any form of insanity through toxæmia or exhaustion of the higher psychical centres, from worry or anxiety, these factors having a modifying influence on the character of the mental symptoms. Why one case of cerebral syphilis should present psychical symptoms and another be free from such complication is best accounted for by the greater vulnerability or "susceptibility of the tissues of the person to disease in general, the one previously constitutionally sound, resisting to the utmost tissue degradation, the other, with inherited tendencies to cell decay, falling a ready victim to the toxin."[3] Exhaustion due to mental stress of any kind, venereal excesses, depressing effects of injury, and intoxications and infections become determining factors in the production of syphilitic insanity, here, in the words of Mickle, playing a more important part than the frequent one of bringing on the sudden outbreak of an already existing but slumbering brain affection.

[1] Paper presented by Dr. Diller before the meeting of the American Neurological Association, held in Boston, 1901.

[2] W. Julius Mickle, M.D., "On Syphilis of the Nervous System," Brain, 1895.

[3] Berkley, Mental Diseases, p. 212.

The psychical symptoms, whether of simple intellectual enfeeblement, mental excitation, delirium, sudden furibund mania, stuporous states, and, more rarely, acute melancholia or persecutory monomania, may at first be free from any association or combination with other cerebral affections of specific origin, such as sudden faints, headache, convulsion, aphonia, facial paralysis, optic neuritis, hemiplegia, or monoplegia. These cases, fortunately rare, present great difficulties in diagnosis, and must frequently be overlooked, the one hope—*i. e.*, early institution of appropriate treatment—thus being lost to the patient. Of some significance, however, are tendencies to remission and to relapse which characterize the psychical as they do the somatic signs of the disease, which phenomena are explained by the underlying pathology. Whether they be of the hypochondriacal, delirious, melancholic, or maniacal type, or various combinations of these, they tend to pass into dementia, with the predominance of negative intellectual symptoms, often associated with failure, weakness or loss of attention, will, memory, with deterioration in moral sense, ideas of propriety, and a general mental degradation.[1]

In the majority of cases, however, the association and commingling of somatic signs characteristic of the luetic process, with psychical symptoms varying with individual cases, reveal the specific nature of the phenomena. Appreciation of the significance of these important criteria is essential if the patient is to have the chance of recovery only afforded by early diagnosis, with the institution of energetic specific treatment. The following case is given in some detail because it illustrates many of the more important diagnostic point by which syphilitic insanity may be recognized:

A. P., an oil driller, aged thirty-five years, was admitted to Dixmont Hospital on December 6, 1900, at which time but a meagre history was obtainable. A sister, who subsequently visited him, gave the following history:

Family History. His father suffered all his life with rheumatism; his fingers were deformed and shoulder crippled. Throughout life he was eccentric, had an uncontrollable temper, and periodically behaved as though quite out of his mind. He died of pneumonia at the age of seventy-nine years. His mother is living, aged seventy-six years. She is considered by strangers to have been queer all her life, and her daughter states that, in her opinion, she is insane at the present time. Two maternal uncles were insane, one being defective from boyhood; in the other insanity developed later in life. Of two brothers, the elder, always defective, was early sent to an institution for the feeble-minded; later he was an inmate of the State Asylum at Harrisburg, and, at the age of seventeen years, of the Western Pennsylvania Hospital at Dixmont. He now makes his home with relatives—a dull, feeble-minded old man, although only fifty-six years of age, barely able to look after

[1] Fournier, quoted by Mickle.

his personal needs. The second brother, aged fifty years, though peculiar from childhood, was of average intelligence and acquired a good education, becoming an artist of fair ability; at least the sale of his paintings brought him a living. Never well-balanced, he would change from one occupation to another, giving up remunerative positions without adequate cause. Dissipated since early youth, he now has locomotor ataxia. He is far from normal mentally, although able to earn enough to pay his board among strangers. A sister, aged thirty-eight years, presents no psychical abnormality, but is markedly neurasthenic.

Personal History. The patient had measles at two years of age, which left him with a chronic diarrhœa lasting a year or more. A few years later the left side of the head and face was affected with a skin disease which his doctor termed "scrofula;" this lasted many months. Up to the age of sixteen years he was subject to violent attacks of intestinal colic. He was always considered a bright and clever boy, with mental attainments quite up to the average (common-school education), but early inclined to all kinds of excesses. He has used large quantities of tobacco and whiskey freely and continuously since he was eighteen years of age, but, like most of his brothers, was profoundly influenced by a small quantity. At twenty-two years of age he married a woman beneath him in social position; living unhappily, he was absent from home for longer or shorter periods, and a consecutive history cannot be obtained. It is known, however, that four years ago he contracted syphilis—a primary sore being followed by sore-throat, alopecia, and skin eruption—for which he has since been continuously under treatment. Although working hard at his occupation of drilling oil wells, he was never compelled to lose a day until an accident occurred which befell him in August, 1899, when several of his ribs were fractured in being struck a glancing blow by a falling clamp which weighed 1000 pounds. Before he completely recovered from the results of this accident he resumed work in a place where malaria was prevalent (epidemic), acquiring the disease, from which he suffered continuously for three months. Now, unable to work, he made his home with his sister, who noticed that he occasionally appeared as though dazed, worrying much about his condition, and at one time he talked of suicide. He complained of constant and intense pain in his head. (It cannot be determined that this was markedly worse at night.) In January, 1900 (three years after he acquired syphilis and eleven months before his admission to the hospital), he became irritable and suspicious, complaining that his mother and sister treated him badly, were not like they used to be, and blamed them for all his misfortunes. About this time he had some trouble with his eyes, of which only a vague description is furnished; but it is known that the left eye was so seriously involved as to lead him to consult a physician. Despite all treatment, vision in this eye grew progressively worse. Leaving the home of his sister, he now returned to his wife, with whom he lived until two months previous to his admission to the hospital. All that can be learned about him during this period is that, although able to work at his trade from time to time, he began to grow dull and stupid and had occasional violent outbursts of temper, in one of which he murderously attacked his wife with a poker. In July he had two fainting spells, in which he was seen to stagger before falling in total unconsciousness. About this time the patient complained of hearing queer noises, which he located "within

his head;" occasionally these were so distinct that he would go to the door of his shop, thinking he heard bands of music. Shortly afterward he was confined to his bed by an attack of "fever, with delirium," during which he saw objects on the wall and talked wildly about "imaginings, which all seemed to be of the horrible order." In this attack stupor alternated with delirium for nine to ten days, when the patient seemed suddenly to grow better, recognizing his surroundings, though slightly confused for several days. One week later he was able to return to work; soon afterward he had a second attack, which was more severe, but recovery took place as in the preceding one. Three weeks later another attack occurred, since which time he has never seemed like his former self. From October 1st to November 20th he again made his home with his sister, who states that he had some trouble with his left arm (lower extremity not involved), and could not use it for several days; but it rapidly cleared up, leaving no trace after the lapse of a couple of weeks. During this time, when not in a dull and stupid condition, he was irritable or sullen.

Examination by Dr. Walker, upon admission, shows him to possess a fairly well-developed musculature and to be well nourished. His skin is healthy, but old scars are seen over the left temporal and right anterior tibial region. There is extreme pallor of the surface of the body. The heart-beat is very feeble; no abnormal sounds. The pulse is 92; irregular. There is no abnormality of the respiratory organs. The tongue is clean, and the digestive functions, apparently, are well performed. Urine: Specific gravity 1024; shows a faint trace of albumin. Microscopical examination was not made. There is no paralysis. The patient is dull and listless; he answers questions in monosyllables, without appreciation of their import. The tendon reflexes are all exaggerated, more markedly upon the left side; no ankle clonus. Superficial reflexes are present and normal. External examination of the eyes reveals nothing abnormal. Pupils: Right, 3 x 3 mm.; left, 3½ x 3½ mm.; margins are regular; irides react promptly to light and accommodation. Ophthalmoscopical examination is made with difficulty, but repeated attempts reveal in the right eye no gross abnormality; in the left, however, is seen deep, atrophic excavation of the disk, presenting the bluish-white appearance of neuritic atrophy.

December 11th (five days after admission). The patient has been dull and confused since admission; to-day he is more restless, muttering in low delirium, and is with difficulty kept in bed.

16th. Confusion and delirium are increasing, with active motor agitation. This morning at 2 o'clock the nurse noted that respiration was very shallow (7 to the minute) and the radial pulse was scarcely perceptible. He revived under strychnine and atropine, given hypodermically. He remained in a stuporous condition for eight to ten hours, from which he could not be aroused to note anything understandingly, followed by active delirium, with sense deceptions of a horrifying character. Marked speech defect is now manifested in stammering and lisping, and the lips are puffed out with each expiration. There is also difficulty in swallowing, attempts to do so bringing on choking spells.

20th. Delirium of varying degree of intensity continued until this morning, when he fell into a sleep of four hours' duration, from which he awakened with inquiry as to his whereabouts. He recognized his sister, who was present, for the first time; and for a period of seven or

eight hours he was conscious of his surroundings to the extent of making his wants known. He then relapsed into a semi-stuporous condition, (urine and feces passed under him), from which, for a time, it was impossible to arouse him to give nourishment and medicine.

23*d*. The stupor deepened into coma, and ended in death, which occurred to-day, seventeen days after admission.

Prominent in the history of this case are the marked hereditary brain instability, further increased by the patient's habits; prolonged exposure of vulnerable tissue to the poisons of tobacco and alcohol, followed by those of the toxins of syphilis, and, later, malaria, with the further depression of the vital forces by traumatism. That the brain disease was hastened in its evolution through the cerebral congestions caused by malaria seems probable. At all events, under the contributive influences of these various factors we see developing in this patient headache, optic neuritis, sudden faints with vertigo, transient and incomplete monoplegia, bulbar symptoms, with the psychical manifestations of mental apathy, depression, irritability, suspicions, violent outbursts of temper, hallucinations, stuporous delirium, and rapidly advancing dementia, this combination of psychic with somatic symptoms presenting the condition to which the name of syphilitic insanity is given.

The autopsy was performed four hours after death by Drs. Walker and Burns. The brain only was examined.

No peculiarity was noted about the skull; the dura mater, however, was somewhat adherent upon the right side. The brain itself, as well as the pia mater covering it, presented a healthy appearance; in short, no pathological changes whatever were visible to the naked eye, save that the convolutional arrangements in several particulars were somewhat atypical. The microscopical examination was made by Drs. Burns and Diller.

Several pieces of the cortex were excised, placed in alcohol, and subsequently stained by the Nissl method. Portions of the cortex, the entire brain stem, and the upper segments of the cord were preserved in Müller's fluid. Sections of them were subsequently made and stained by the Weigert, Pal, and Van Gieson methods; and subsequently, at the suggestion of Dr. W. G. Spiller, other sections were stained with Delafield's hæmatoxylin.

The sections stained by the Nissl method showed the cells of the cortex to be normal in the main, although here and there a slight granular degeneration of the cytoplasm or a dislocated nucleus was noted. The vessels were best seen in the sections stained with Delafield's hæmatoxylin. The vascular changes were particularly studied as occurring in the pia mater, the cerebral peduncles, the interpeduncular space, and the oblongata. While a large number of vessels were seen exhibiting little or no change, the majority of the vessels showed more or less change. This change, for the most part, consisted in the proliferation of small round-cells from the adventitia into the perivascular lymph channel, partly or wholly obliterating that passage-way. (See Fig. 4.) In others the proliferation, however, was in both the middle and ex-

ternal coats of the vessel. (See Fig. 3.) In a less number of cases there was proliferation into the lumen of the vessel, which was only

FIG. 1.

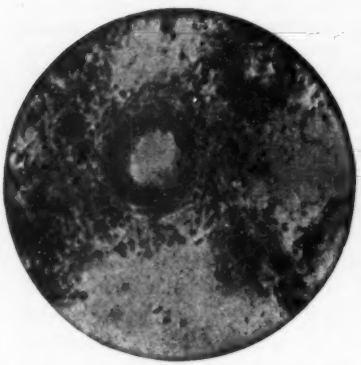

FIG. 2.

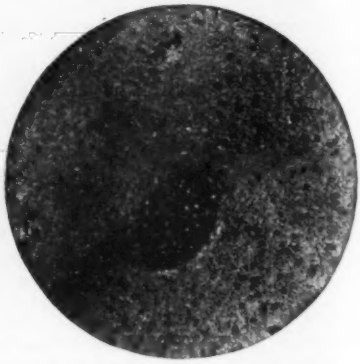

Fig. 1.—The infiltration involves the adventitia and perivascular lymph space, springing apparently almost wholly from the former.

Fig. 2.—The infiltration here is into the lumen, occluding the same, and due to proliferation from the intima.

FIG. 3.

FIG. 4.

Fig. 3.—An enormous mass of round-cells is seen involving all three coats of the vessel and filling the perivascular lymph space, and extending far beyond its limits. The lumen of the vessel, closely packed with blood-cells, is but little occluded by the infiltrate. Here it seems probable that the adventitia was the starting-point of the proliferation.

Fig. 4.—The infiltration here involves all three coats of the vessel and occludes completely both its lumen and that of the perivascular lymph space as well, being pretty well limited within the boundaries of the latter.

rarely found to be entirely obliterated by the proliferation (see Fig. 2), while in a still less number of instances the infiltration occluded both the lumen and the perivascular lymph space and obscured the

entire wall of the vessel. (See Fig. 4.) In a few instances the lumina of the vessels were greatly narrowed by an overgrowth of the media rather than by round-cell infiltration.

The facts that the nuclear proliferation was seen most frequently involving the adventitia, that the media was next most frequently involved, and that proliferation from the intima into the lumen alone was seldom seen, lends support to the view expressed by Berkley,[1] that the source of the proliferation is chiefly, or most frequently if not always, from the outermost coat of the artery, as against the views of Heubner and other earlier writers. Indeed, our sections correspond closely to those shown in Figs. 1, 2, and 3, Plate 4, in Berkley's text-book.[2]

Thus it will be seen that through the changes emanating from the vessel walls the flow of lymph in the perivascular channels must have been greatly interfered with, and that this was probably a much more powerful factor in the production of the symptoms observed than the narrowing or occlusion of the lumina of the vessels.

The second case which we shall report is one which came under the observation of one of us (Dr. Diller) at the Allegheny General Hospital. It is introduced in this connection because it was also diagnosed as a case of syphilitic insanity, whereas the autopsy demonstrated it to be one of paralytic dementia. It will illustrate the great difficulty, at times, of making a differential diagnosis between these two affections.

W. M. H., aged thirty-three years, white, male; married; a machinist, but he had also been a sailor for several years.

Family History. Entirely negative.

Previous History. He had always been in excellent health previous to February, 1900, with the exception of a varicocele, which he has had for twelve years, and which he says followed an injury received while a sailor. It caused him a great deal of trouble, which was, however, mental rather than physical. He has always been a hard worker. No history of syphilis was obtainable.

History of Present Condition. On February 17, 1900, while in his usual health, he had an attack in which the left side of the body became numb, with absolute loss of power on the same side. There was no loss of consciousness, and the patient recovered completely in from five to seven minutes. He had a similar attack two weeks later. In April two more attacks occurred, and another in July. Following the attacks in April his wife noticed impairment of memory. This was the first mental symptom observed.

On the morning of October 8, 1900, while crossing the room, he fell to the floor unconscious, and was completely paralyzed on the left side. He remained in this condition all day. During the following night, about twelve hours later, he had three general convulsions, lasting in all about half an hour. On the following day, October 9th, the paralysis was less marked, but he could, however, be aroused. On October 10th he had many delusions, and was thought to be temporarily insane. Three days later, on account of these mental symptoms, he was taken to St. Francis' Hospital, where he remained three weeks, and was then

[1] Mental Diseases, pp. 90, 91. [2] Op. cit.

discharged as cured. From this time until his admission to the Allegheny General Hospital, on December 10, 1900, he seemed to be "childish," and had a good deal of difficulty in utterance. In health he had always been a fast talker, but following these attacks he spoke very slowly. He had no headache or vertigo, and never vomited. His eyesight was good.

He came to the Allegheny General Hospital on December 10, 1900, for an operation for varicocele. His mind was constantly fixed on this condition, and it was thought that perhaps an operation would help him. The operation was performed the following day by Dr. R. L. McGrew, and the patient made a good recovery. On December 20th he was about to leave the hospital, when he was suddenly seized with a severe general convulsion, lasting about fifteen minutes. He had a second one that night, and a third the following morning (December 21st). These attacks left him in a condition of hebetude, with delirium. There was paralysis of his entire left side, with partial paralysis of the muscles of speech. His eyes were turned constantly to the right, but he could move them to the left. The pupils were widely dilated, the left more than the right. The left pupil reacted moderately to light, the right scarcely at all. Knee-jerks: Normal on the right, exaggerated on the left. Ankle clonus is marked on the left, absent on the right side. Babinski sign is absent.

December 23d. Paralysis of the left arm is almost complete; paralysis of the leg is much less complete. No change in the knee-jerks. Ankle clonus is not present. The mental condition is difficult to determine exactly, but seems to be one of dementia, with stupor. Speech is paralytic.

26th. Mental condition is much improved. Left arm and leg are much stronger. Conjugate deviation has entirely disappeared.

27th. The eye-grounds were examined by Dr. Duncan and found to be normal.

31st. He moves the left side very well to-day. Pupils are equal and react well to light. The patient was discharged immensely improved in every way. For a few days after his discharge from the hospital he was able to go about, and seemed pretty well, but there was an evident dementia, and the speech strongly suggested paralytic dementia. He was seized with another convulsion at home, with left-sided paralysis, also with delirium and stupor. On January 10, 1901, he was taken to "Claremont." He improved very rapidly for several days, when he had another severe convulsion, followed by left-sided paralysis and great mental confusion. This partially cleared up, and he continued to improve for several weeks, until March 9th, when death occurred, following a very severe convulsion.

The autopsy was performed twelve hours after death by Dr. Charles E. Ziegler, of the Allegheny General Hospital, and Dr. Diller, at the City Poor Farm at Claremont, the brain and upper portion of the cord alone being removed.

The calvarium was very thick, and the dura itself much thickened, was strongly adherent to it. The pia was watery, œdematous, and of a milky-white opacity over the parietal and frontal lobes, while that over the occipital and temporal lobes, as well as that over the base, presented a comparatively healthy appearance. There was a subpial hemorrhage at the top of the Rolandic region on the right side, another

a half-inch anteriorly, and a third the same distance behind. There were also subpial hemorrhages at the anterior extremities of both frontal lobes. The vessels were large and tortuous over the frontal and parietal lobes. The convolutions could not be made out because of the opacity of the pia. The pia was stripped off with difficulty, small portions of the cortex adhering to it, leaving a roughened appearance of the cortex behind. In short, the naked-eye picture was typically that of paralytic dementia.

Small pieces of the cortex were placed in alcohol, and sections from these were afterward stained by the Nissl method, while the brain stem and pieces of the cortex were placed in Müller's fluid for preparation by the Van Gieson, Weigert and Pal methods. The microscopical examinations were made by Drs. Burns and Diller.

There was greatly increased vascularity everywhere throughout the cortex. There was a large amount of cellular exudate in the lymph spaces. The vessels were, as a rule, dilated, frequently to several times their normal size.

The great majority of the cortical cells showed no changes, but in a large number various morbid changes were seen. A number of cells were seen showing granular degeneration of the cytoplasm, most of which contained a healthy nucleus; but in some of them the nucleus was indistinct or irregular in outline, while in still others it had wholly disappeared, leaving only a mass of granular detritus behind it. In a few cases only faint skeleton outlines of the cell were to be observed, in some of which the nucleus could still be seen. In many cells a granular degeneration had involved one portion only of the cytoplasm. In a few cases the nucleus was shrunken and irregular in outline. A dislocation of the nucleus was observed frequently both in degenerated cells and in cells which otherwise appeared normal. The nucleus was found at the side of the cell most frequently, sometimes protruding beyond the side. In certain areas of the cortex there was great scarcity of cells, and it seemed that here cells had probably disappeared entirely.

Thus it will be seen that changes characteristic of paralytic dementia were discovered both by the naked eye and the microscope. That the case was one of this disease we do not doubt.

The clinical recital of the case will alone be enough to suggest the difficulties of diagnosis. Indeed, the case so strongly suggested syphilitic insanity that it is difficult to see how a correct diagnosis, at least in the earlier development, could have been made from the symptoms. The recurrent attacks of hemiplegia, convulsions, mental confusion, stupor, maniacal excitement, with the rapid recovery, in a thirty-five-year-old-man, seemed to point to syphilis as the cause of the symptoms, in spite of the fact that dementia and paralytic speech were present.

When the man was first seen by Dr. Diller, however, in reply to the question, "How are you?" the answer, "V-e-r-y w-e-l-l," was made in the way so highly characteristic of paralytic dementia that the thought at once arose that the patient was a case of this disease. After leaving the Allegheny General Hospital, when he had very largely recovered, a moderate dementia and this same characteristic speech were both

noted, and the suspicion still lurked, although the evidence seemed greatly in favor of the diagnosis of cerebral syphilis. With the subsequent history as it now stands, however, it must be admitted that the correct diagnosis might be made.

For the excellent photomicrographs which illustrate this paper we are indebted to Dr. A. Letèvé, of the Magee Pathological Institute of the Mercy Hospital.

A CASE OF MYXŒDEMA, WITH A STUDY OF THE BLOOD AND URINE DURING TREATMENT.

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The following study of a case of myxœdema may not be uninteresting in connection with the views respecting the exact function of the thyroid gland. The broad symptomatology, indicating as it does a disturbed condition of the various organs of the body, tends to some confusion in the interpretation of functions. Many of the symptoms of myxœdema are merely secondary, and many of the so-called functions of the thyroid gland are very secondary in character. That the chief function of the thyroid gland, however, has to do with the matter of the destruction or elimination of some substances which are prejudicial to the body, and which, if retained, produce various symptoms of an intoxication of peculiar type, with an ultimately fatal result if untreated, is accepted by most clinicians, and this view is amply supported by clinical and experimental facts.

On the morning of May 22, 1900, I was called to see Mrs. F., aged forty-three years, married, mother of seven children. Present illness dates from about fifteen years ago, when she began rapidly to gain flesh, having previously weighed about 110 pounds. Periods of occasional fainting, with marked irritability of temper, characterized her condition up to about five years ago. The gain in flesh continued without intermission, and during the winter of 1895 and subsequently she suffered intensely from the cold. At about the same time the skin began to show unusual dryness and marked thickening in some portions, especially the scalp, with areas of yellowish discoloration, and the hair lost its soft texture, became dry and brittle, and began to fall out. The increase in flesh continued; the abdomen became pendulous, and the limbs puffed with a solid œdema, which tended to become more marked toward evening. Some puffiness about the eyes was also present, and a certain amount of deafness was noticed by members of the family. Patient was exceedingly irritable and was easily angered. On slight provocation passed into periods of unconsciousness. These were occasionally preceded by a period of motor excitement characterized by acts of violence. Latterly these acts of violence have been more infrequent.

At the present time she complains especially of inability to concentrate her attention or to accomplish anything. Little difficulties appear

insurmountable, and the ordinary requirements of living, such as bathing, dressing, walking, and riding, require great effort. Walking is done with some difficulty. In fact, for some time patient has confined her walking to the house, and experiences great difficulty in getting up and down stairs. Before the last child was born, about sixteen months ago, there was great swelling of the limbs and general discomfort, but the labor was uncomplicated and uneventful. For the past five years, but more especially during the past two years, she has suffered from a severe type of metrorrhagia. Some giddiness and pain in the head are experienced when standing erect, and latterly she has been rising late in the morning and taking but little physical exercise during the day. At present she is unable to do anything requiring effort, takes little interest in her family, household, and social affairs, and dreads and avoids all physical exertion. It is with great difficulty that she manages to take a carriage ride daily.

Examination showed a peculiar solid œdema over the whole body, especially marked in the extremities; skin very dry, thick, and scaly, and of yellowish cast; scalp thick, dry, and very scaly; hair dry and very thin; eyebrows thin. There were marked pockets beneath the eyes and some puffiness of the upper eyelids. The nails were thick and brittle; voice heavy and husky; hearing very poor; sight poor; many small warts about the neck and hands; marked fulness in both supraclavicular regions, and the thyroid gland appeared atrophied. Heart very slow and a little irregular; sounds weak and greatly muffled, no murmur; abdomen large and pendulous; bowels constipated; appetite poor; gait very unsteady and tottering. Mentally she evinces a poor memory, especially as regards recent events, and is very dull and apathetic, indifferent and markedly suspicious. Temperature 98.6° F.; pulse 57; weight 201 pounds. Patient wears spectacles, but states that they are no help to her, notwithstanding the fact that they have recently been fitted.

Urinalysis showed the presence of albumose and a trace of serum albumin. Urine pale, specific gravity 1007. The sediment presented an occasional cylindroid and many epithelial cells. Urea excreted in twenty-four hours, 16 grammes. The blood examination showed red blood-cells 4,293,000, and white blood-cells 7200 per c.mm.

On May 23d and 24th patient took three grains of desiccated thyroid two hours after breakfast. Another three-grain powder was added on the 25th and subsequently, so that the patient received six grains of thyroid daily. This amount was not increased for several weeks because of the effect on the heart. Until May 31st the patient continued to spend most of the time in bed, and subsequent to this date was kept in bed continuously. The pulse gradually increased in frequency until on the evening of May 31st it was 74 and fuller and stronger. Mentally she was very much better and more cheerful than for many months, and a "stuffy feeling" of which she had complained for a long time had entirely disappeared. She seemed especially elated over a return of her hearing, and repressed members of her family who continued to deem it necessary to converse in a loud tone of voice. Weight, May 31st, 185½ pounds. Blood examination was made on June 1st, with the following result: Red blood-cells, 3,520,000; white blood-cells, 4500. The urinalysis still showed a trace of albumin. The urea excreted rose on June 3d to thirty-one grammes, and on the 4th was twenty-seven grammes. Patient continued to take six grains of thyroid

daily throughout the month of June, and showed marked improvement in all her symptoms. Once the thyroid was discontinued on account of a rapid and compressible pulse, ranging from 90 to 100, but these symptoms subsided in three days, and the thyroid was again resumed. The weight on June 10th had fallen to 177½ pounds, and on June 17th to 176 pounds. The skin showed marked improvement over the whole body, and she heard so well that the noises of the street were troublesome, notwithstanding the surroundings of a quiet country home. The eyesight was greatly improved, and she read without glasses. On the 12th and 13th patient suffered considerably with ovarian neuralgia, which she said resembled very closely some similar periods during her girlhood. This ceased on the 14th, and did not return. The heart-sounds, which were very weak in the beginning of the treatment, became firmer and better punctuated as it progressed. The skin of the face improved in color and texture, and the pockets beneath the eyes rapidly disappeared. The June menstrual period was normal, for the first time in many years, and latterly the bowels have moved without a cathartic and are very regular.

During the month of July the general improvement continued. Patient suffered from occasional periods of faintness after slight exertion, but for the most part remained in bed and was very comfortable. Weight on July 8th was 173 pounds, on July 15th, 171 pounds, on July 22d, 168 pounds, and on July 29th, 166 pounds. The month was eventful in that the patient suffered from functional disturbance of the liver, with diarrhœa, and neuralgic pains in various portions of the body at irregular intervals. The urine still showed a trace of albumin, but no casts, and the amount of urea ranged from 25 to 30 grammes daily while thyroid was being given. Albumose was not detected after the first urinalysis. During the periods of illness above noted the thyroid was discontinued for a brief time and the urea dropped immediately; thus on the 13th, 14th, 15th, and 16th the daily amount of urea excreted was respectively 18, 14, 24, and 20 grammes. The rise on the latter two days was probably due to the administration of certain hepatic stimulants. The action of thyroid in increasing the daily amount of urea has been noted by a number of authors. In this case it was very striking, falling regularly during its omission and rising to the normal amount during its administration. This would seem to warrant placing the thyroid substance among the direct hepatic stimulants.

The blood examinations are presented below in tabular form. The examination on May 22d was preliminary to the administration of thyroid; all the others were done during treatment.

Date.	R. B. cells.	W. B. cells.	Lymphocytes.	Large mononuclear leucocytes.	Neutrophiles.	Eosinophiles.
May 22 . . .	4,293,000	7,200	25.6 per ct.	8.2 per ct.	63.4 per ct.	2.6 per ct.
June 4 . . .	3,520,000	4,500	16.8 "	4.0 "	78.2 "	1.0 "
" 11 . . .	3,872,000	5,400	28.6 "	6.4 "	64.4 "	0.6 "
July 14 . . .	4,384,000	6,000	18.0 "	9.4 "	71.0 "	1.6 "
Aug. 8 . . .	4,704,000	6,266	21.6 "	7.2 "	69.4 "	1.8 "

The blood examinations were all made between 11 and 12 o'clock in the morning, about four hours after the patient had eaten breakfast. In every instance 500 cells were counted in making the differential count.

There are several points of interest in the above table. The preliminary blood examination on May 22d showed 4,293,000 red blood-cells, and on June 4th, 3,520,000 red blood-cells per c.mm., a difference of 773,000. I attribute this apparent diminution in the number of red blood-cells to the increased activity in the various organs of the body. As a result, the fluid elements of the blood were greatly increased, while the corpuscular elements remained stationary. In other words, the apparent diminution in the number of the red blood-cells was due to the increase in the fluid elements of the blood consequent upon the pouring into it of fluids from the various organs which were recently awakened into activity. These facts tend to negative the view that the thyroid gland is directly a blood-forming organ. Under the conditions here presented it is hardly conceivable that the thyroid, if a direct blood-forming organ, would thus fail primarily to exhibit this property. The subsequent steady increase in the number of the red blood-cells is to be noted.

The drop in the number of white blood-cells per c.cm. at the second examination is to be attributed to the same cause, and their subsequent steady increase is to be noted.

The differential count showed nothing distinctive aside, perhaps, from the primary drop in the number of eosinophile cells and their subsequent gradual increase in number. A feature of some interest was the large size of the blood platelets. Many approached the size of a red blood-cell. They had attained to about their normal size three weeks after the beginning of thyroid administration. The hæmoglobin was found somewhat diminished at the preliminary examination, and there was considerable irregularity in the size and shape of the red blood-cells. This condition improved steadily, and at the examination on August 8th the cells were nearly normal.

The albuminuria, which was fairly well marked in the beginning, disappeared completely with the improvement in general symptoms.

The lowest weight attained was 166 pounds, and was recorded on July 29th. During the beginning of September the patient began to sit up regularly, after having spent three months in bed.

The largest amount of thyroid taken while the patient was in bed was nine grains daily, during the last two weeks of August. Notwithstanding this the patient gained some in flesh (two pounds) and evinced an excellent appetite. The hair grew rapidly, and during the first three months attained considerable length. The skin of the scalp presented a perfectly normal appearance. About the middle of September the patient began to go about, and was soon able to resume her normal place in her family. A year has now passed, during which she has been very well.

She is now taking regularly ten grains of thyroid daily. During the last winter she took as high as fifteen grains daily and suffered no ill effects. In fact, this amount seemed necessary in order to maintain a state of health. It has been my custom to reduce the daily amount of thyroid when the heart shows its effect in a rapid and compressible pulse. Under these circumstances I have found digitalin useful.

Patient's general condition is excellent at the present time. The pulse, which a year ago was weak and poor, is now strong, full, and regular.

I am of the opinion that it is a mistake to crowd the thyroid in the beginning of treatment. The heart muscle, from a long-continued absence of the thyroid secretion, is so poor and weak that it stands its primary depressing effect badly. When a little time has elapsed, and the heart has again become accustomed to the effect of the thyroid substance, it may be increased with propriety and benefit.

A FATAL CASE OF ACETANILID POISONING.

By Philip King Brown, M.D.,
INSTRUCTOR IN CLINICAL PATHOLOGY AND MEDICINE, COOPER MEDICAL COLLEGE, SAN FRANCISCO, CALIFORNIA.

B. K., male, aged thirty-seven years, shoemaker, was brought to the Mount Zion Hospital at 5 P.M., January 22, 1897. The history of the case was as follows:

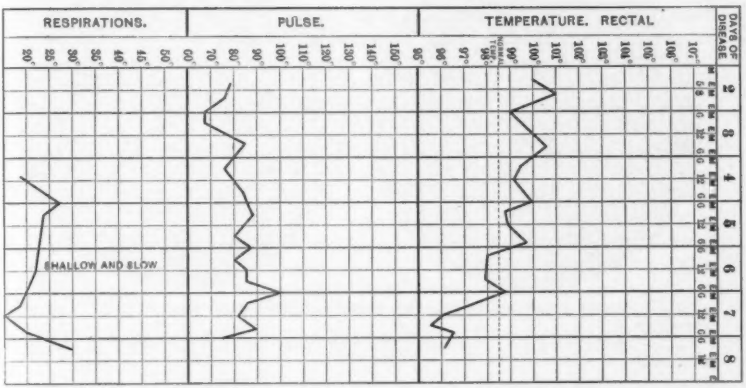
On the morning previous the patient was found by his attending physician slightly delirious and complaining very much of his head and of a pain in the left umbilical region. The temperature was much elevated, the heart rapid, and there was marked constipation, slight jaundice, nausea, and some vomiting. The day before the patient had had one chill. Calomel in small doses, followed by salts, gave a copious but bloody movement of the bowels. The urine, which was clear on the previous day, became dark red. No facts could be obtained from the family which threw any light on the case, and the patient was sent to the hospital, where I saw him an hour after his admission. It was ascertained later that a lodge physician had given him sixty grains of acetanilid in six powders for his headache, and the patient had taken them all within a few hours before his regular attending physician was called.

At the time of entrance the pulse was 78, soft, and easily compressed; the temperature was 100.2° F.; the lips and nails were extremely cyanotic, and there was slight jaundice over the whole body. The heart-sounds were normal and the action fairly strong; respirations shallow and somewhat increased; occasionally a slight, harsh cough. Lungs and spleen normal; liver somewhat enlarged, but not painful to pressure. Abdomen slightly distended in the region of the descending colon; no fecal impaction felt. Patient complained of a slight pain over the whole left side of the abdomen, and was sensitive especially in the epigastric region and over the region of the left kidney. Skin moist, tongue not coated, gums bluish, no hemorrhages into mucous membranes. The urine, 300 c.c. of which were passed shortly after entrance, was strongly alkaline and of a deep red color, nearly black. The color of the filtrate was unchanged; the sediment was small in amount, and contained granular casts, small and medium sized. The granules were fine, and ranged from golden yellow to deep brown. There were a few granular casts with epithelial cells attached. No leucin or tyrosin found. The specific

gravity was 1016, and there was a large coagulum on boiling. No biliary coloring-matter. There was complete suppression of urine after the second day in the hospital. Only 150 c.c. were passed that day, and the character of it was the same as that first passed. Chemically and by subsequent spectroscopic examination the coloring-matter proved not to be hæmoglobin, but hæmatoporphyrin.

Six days later the patient died. In the meantime every effort was made to maintain the functions of all the organs. The great thirst was quenched with milk when the patient's stomach would stand it, but very soon he vomited all that was given to him and he had to be fed by the rectum. The stomach continued sensitive to pressure and to food to the end, and, in spite of the fact that nothing was given by the stomach for days, vomiting continued. The vomitus was mostly a greenish mucus. The cough and expectoration were continuous, the expectoration consisting chiefly of a clear, sago-like mucus.

Throughout the week the patient was slightly delirious and wholly indifferent to all that went on around him. The reaction of his pupils



to light grew more and more sluggish, and in the end the pupils were greatly dilated. Hyperæsthesia was especially marked over the abdomen, but disappeared with continuous manipulation, to return if the patient were quiet a few minutes. The reflexes were exaggerated for the first few days, and then gradually disappeared. The exaggeration, however, occurred only at the first few elicitations at each trial.

The extremities were constantly cold, although external heat kept up the body temperature fairly well. The temperature fell slowly to normal on the fourth day, and was subnormal thereafter, reaching 95.5° F. by the rectum on the evening before death. (See chart.) The patient's restlessness steadily increased. There was alternate constipation and diarrhœa, and forty-eight hours before death and twenty-seven hours after the suppression of urine there began to be constantly blood coloring-matter and broken-down blood cells in the feces, which had until then been blood-stained only occasionally.

The heart began to fail on the fourth day in spite of increasing stimulation. The skin grew more jaundiced throughout the course of the

illness, and by the third day was very dry, as were also the mucous membranes. The respirations, which were slightly increased at the time of entrance, diminished slowly to normal, although they were very shallow, the diaphragm not descending noticeably.

On the sixth day in the hospital two movements of dark red fluid were followed shortly by the passage of sheets of mucous membrane several square inches in size, and two large clots of blood, together about the size of a placenta and stained yellowish-brown on the exposed surface. The distention and sensitiveness in the left inguinal region disappeared after this, but the patient was greatly prostrated and seemed to have some pain all over the abdomen. The thighs were constantly flexed on the abdomen, and the expression of the face grew more anxious. The patient sank rapidly after this, and died eight hours later.

Fig. 1. Fig. 2.

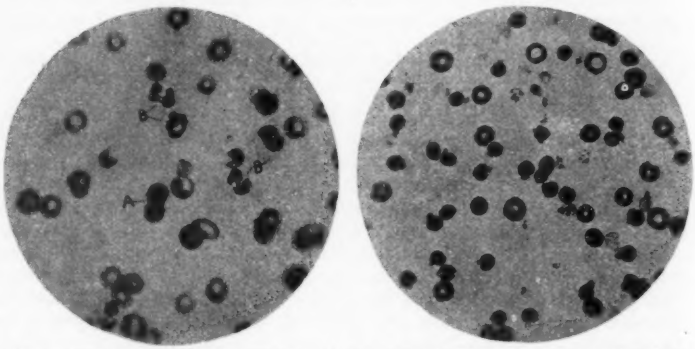


Fig. 1.—Anæmia from blood destruction. A. Red corpuscle in mitosis. B, B. Polynuclear leucocytes.

Fig. 2.—Anæmia from blood destruction, showing direct action of the poison on the red corpuscles.

The superficial circulation was so poor from the start that no attempt at hypodermic medication was tried after the first day. Salt solution per rectum was well retained at first, and was to have been tried intravenously on the day the patient died. Oxygen was not used.

Blood examinations were made on four days, with the following results:

On the fourth day of illness, the second day in the hospital, stained cover-slip preparations showed a destructive process to the red cells and a great increase in nucleated elements, most marked in the nucleated red cells. (Fig. 1.)

The following are the blood-counts and hæmoglobin estimates:

	Hæmoglobin.[1]	*Erythrocytes.*	*Leucocytes.*	*Nucleated red cells.*
5th day of illness,	40 per cent.	2,200,000	62,400	17,600
6th " "	35 "	1,490,000	62,935	25,705
7th " "	33 "	1,400.000	51,400	19,000
8th " "	32 "	1,166 000	66,450	22,150

[1] By von Kalden's method.

DIFFERENTIAL COUNTS.

	Nucleated red cells. (Seen during a count of 500 leucocytes.)			Leucocytes.			
	Megaloblasts.	Normoblasts.	Microblasts.	Polymorphonuclear.	Large lymphocytes.	Small lymphocytes.	Eosinophiles.
5th day	39	60	1	70 per ct.	26 per ct.	4 per ct.	0
6th day	15	73	12	59 "	30 "	9 "	2
7th day	...	...	...	...	...	...	...
8th day	19	65	16	57 "	35.6 "	6.5 "	0

Among the large lymphocytes I have included a considerable percentage of myelocytes.

The relation of the nucleated red cells to white cells increased from a proportion of 1 to 5 on the fifth day to 1 to 3 on the eighth day, and it seemed from the active karyolysis and karyokinesis of the nucleated red cells that every effort was being made by nature to increase the number of red corpuscles.

The specimen of the fifth day (see Fig. 2) shows the direct action of the poison on the red cells. They are of all sizes and shapes, the microcytes showing an unusual sort of distortion, unlike crenation or the changes in microcytes of pernicious anæmia, and the megalocytes having no distortion and resembling strikingly those of pernicious anæmia. Indeed, as the process advanced the picture of the red cells grew more and more like pernicious anæmia. The average diameter of the cells increased, the microcytes grew fewer in number and showed less poikilocytosis, if the distortion could be called that, and the color index increased from about one to considerably above it.[1]

That the poison acted directly on the cell membranes as well as the protoplasm is indicated by the large number of free nuclei of both the red and white cells, those of the red cells being of all sizes.

CONCLUSIONS. The patient's life was sustained eight days after taking sixty grains of acetanilid. There was a general hyperæsthesia and not an anæsthesia, as is stated to result from a poisonous dose of acetanilid. There were increased reflex actions, and motor and sensory functions were retained practically to the end, which is also at variance with the generally accepted result. The patient retained nothing on the stomach from the first, and was fed per rectum. Vomiting occurred frequently, nevertheless. Arterial pressure was constantly low, and the pulse-rate steadily increased until just before death. The respirations were slightly increased, and varied but little until the last few hours, when they increased to 30 per minute. They were always shallow, and the diaphragm did not appear to move at all. The temperature per rectum appears to have been elevated by the toxic dose,

[1] Compare the results obtained by Rosenqvist in animal experiments ("Blutgiftanæmien," Helsingfors, 1900).

and it fell gradually to several degrees below normal. It was 96.5° F. at the time of death, and had been 95.5° F. on the day previous. There was a progressive destruction of red blood-corpuscles down to 1,116,000, with 30 per cent hæmoglobin on the day of death. There was a very marked leucocytosis and a large number of nucleated red cells of all sizes. The alkalinity of the blood (Quinan's method) was diminished 80 per cent. There was hæmatoporphyrinuria, beginning twenty-four hours after the dose was taken, and complete suppression of urine three days before death. Acute diffuse nephritis. Hemorrhage from the intestines. Acute progressive jaundice. Whether the symptoms were aggravated by the calomel administered for constipation I have not been able to determine by animal experiment. The antagonism between antipyrin and calomel is well known, and possibly the closely allied chemical products may also share the antagonism.

Autopsy, Eight Hours After Death. Inspection of opened abdomen showed liver edge sharp, and the organ lying as felt before death. Stomach, normal size. No adhesions anywhere. Bowels and bladder empty. Spleen slightly enlarged, firm, capsule slightly cloudy, trabeculæ clearly outlined, pulp firm, color brownish-red. Liver, normal shape, edge firm and sharp, whole organ hard. Lobules fairly seen, color dark yellowish-brown. Gall-bladder distended with dark greenish-black viscid bile, consistency of melted tar. Left kidney normal size, firm, red-brown color, capsule slightly adherent; no distinction on section between cortex and pyramids. Right kidney slightly larger, granular, and swollen. Heart normal size, right ventricle full of light yellowish-brown fibrin clots. Valves normal. Muscle very yellow. Papillary muscles very yellow; the appearance of fatty degeneration here was very marked. Left ventricle wall 2.8 cm. thick. In both auricles and ventricles, in addition to the large pale yellowish fibrin clots, were small dark red, friable clots the size of peas. Aorta a deep yellow color, slight beginning atheroma at right coronary and in aortic arch, one segment of valve thickened but competent. Tricuspid valve slightly thickened at insertion of chordæ. Brain negative. Lungs, small amount of clear fluid in pleural cavities. Right lung adherent to chest wall throughout entire extent. The adhesions were old, and the lung was removed only by detaching the pleural membrane from the chest wall. No crepitation, but a large amount of bloody and frothy fluid could be squeezed from cut surface on pressure. Left lung presented the same appearances; few old pleuritic adhesions along the anterior base of upper lobe; old puckered scar in apex, surrounded by small area of emphysema. Bladder empty and greatly contracted. Stomach empty; inner surface covered with thick mucus closely adherent to wall; pylorus small, 3 cm. in circumference. Intestines showed chronic catarrh. The thickened mucous membrane was covered throughout with a dark green, thick, closely adherent mucus. The wall itself was discolored, but there was no sign of recent or old hemorrhage anywhere in the intestinal tract or in the submucous or serous layers—in fact, nothing to account directly for blood coloring-matter in the stools. The glands were not enlarged in any part.

LIGATION OF THE COMMON CAROTID ARTERY FOR HEMORRHAGE FROM THE CHEEK; HEMIPLEGIA; RECURRENCE OF BLEEDING; DEATH.

By FRANCIS T. STEWART, M.D.,
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L. R., aged thirty-nine years, laborer, was admitted to Dr. Lewis W. Steinbach's service at the Polyclinic Hospital, July 9, 1901, and the following history elicited: On July 4th, while intoxicated, he received a blow on the left cheek from a fist. July 9th he applied to a neighboring hospital because of repeated slight hemorrhages from the mouth. The same evening he entered the Polyclinic with profuse bleeding from the inside of the left cheek; this was checked with hemostatic forceps and pressure. The bleeding recurred, and ligatures were passed through the cheek around the lacerated mucous membrane, but their good effect was only temporary. Ligation of the facial artery caused a respite, but bleeding again began and pressure was applied. In the absence of Dr. Steinbach I was asked to see the patient July 12th. He was very pale, the temperature 100° F., respirations 40, and the pulse 140, small and easily obliterated. On the inside of the left cheek was a sloughing area about one and a half inches in diameter, opposite the molar teeth. No history of purpura, hæmophilia, jaundice, scurvy or rheumatism could be obtained. Since admission he has bled profusely eight times, each time filling an ordinary pus basin and saturating his mattress; during the last twelve hours bleeding has been constant.

As soon as preparations could be made the left common carotid artery was tied just below its bifurcation. There was no hemorrhage, the operation resembling a dissection on the cadaver. He was liberally stimulated with strychnine and atropine, and about one quart of salt solution introduced into his circulation.

When consciousness returned, twelve hours later, mental confusion, difficulty in talking, and slight impairment of motion in the right face, arm, and leg were noticed. The following day the temperature was 101° F., the respirations 24, the pulse 106, stronger and more full. The paralyses were less marked and the mind clearer. The improvement continued until July 14th, two days after operation, when the bleeding recurred, the patient losing six or eight ounces of blood. The pulse quickly became imperceptible, and the temperature rapidly ascended to 105° just before death.

The autopsy was made by Dr. David Riesman. Rigor mortis was slight, post mortem lividity considerable; the body very pale. The muscles were dark maroon in color. The liver was normal in size, slightly fatty, and showed no cirrhotic changes. The spleen was enlarged, pale, and soft; its weight was 28 grammes. The remaining abdominal organs were normal, excepting a few old adhesions on the right side. The right kidney weighed 200 grammes, had an easily stripping capsule, a diminished cortex, and a variegated color, yellow predominating. The left kidney weighed 120 grammes and presented a small cyst on its surface.

The heart, soft, pale, and flabby, weighed 500 grammes, and contained on either side a chicken-fat clot. The tricuspid orifice admitted three fingers, the aortic and pulmonary valves were normal. The ventricles, especially the left, were dilated; the left wall measured 15 mm., the right 7 mm.

There were numerous adhesions in the pleural sacs, the lungs were emphysematous, and opposite the sixth intercostal space under the pleura were two lipomata the size of ox-heart cherries.

The brain showed marked softening, but no other abnormality.

No thrombus was found in the carotid artery on either side of the ligature. Baumgarten has demonstrated that in the absence of infection no thrombus results after ligation. The bifurcation was about one inch above its usual situation.

The cause of death was anæmia and fatty degeneration of the heart.

The persistent bleeding must have been due to infection, as an interval of five days elapsed between the reception of the blow and the onset of hemorrhage. In a similar case I should first excise the sloughing area and approximate the healthy edges of the resulting wound with sutures.

Of 789 ligations of the common carotid in Wyeth's table 41 per cent. died. This mortality is probably excessive at the present day, for of 47 cases treated by modern surgical principles but one perished (Reyher and Weljaminaw). Cerebral involvement is common, 20 per cent. (Warren), 26 per cent. (Zimmerman), 25 per cent. (Riese). Recurrence of bleeding sometimes occurs owing to the free anastomosis with the vessels of the opposite side.

REPORT OF A CASE OF TETANUS.

By J. Norman Henry, M.D.,
of Philadelphia.

A. C., aged nineteen years, a plumber, was admitted to the Pennsylvania Hospital on July 23d, with the following history:

Fifteen days previously, while walking about in a stable, he ran a nail into his left foot for a distance of about a quarter of an inch. The wound bled very little, but caused him considerable pain for several hours, and remained sore for three or four days. The patient applied poultices, which were followed by considerable discharge from the wound.

Four days later, while in an outdoor shed which had no floor, he stepped on a rusty nail, which ran through his shoe and punctured the skin, but did not penetrate below it. He applied poultices to this also, and the puncture soon healed, after discharging some pus.

Three days before admission, on waking, patient noticed that his jaw was quite stiff, but paid little attention to it. On the day before admission the stiffness of the jaw greatly increased, and stiffness also appeared in the neck and back, which later in the day became very painful, with a sensation of "drawing up," occurring in paroxysms every few min-

utes. A physician was called, who advised immediate removal to a hospital. On admission the following conditions were present:

The patient was a very muscular and robust young man, evidently suffering severe pain at intervals of about a minute. Some opisthotonus present. Skin heavily beaded with perspiration. Pupils equal and react to light and accommodation. Tongue can be fairly protruded, and jaws opened so that three-quarters of an inch space appears between the central incisors.

Temperature 99.6° F., respiration 28, pulse 100 and of good volume. Heart and lungs negative. Muscles of abdomen and back tense and firm, seemingly in state of tonic contraction. A small punctured wound, recently healed, is visible on the sole of each foot. There is no inflammatory zone surrounding them.

All reflexes are intensely exaggerated, and tapping the patellar tendon causes a clonic spasm. The urine was amber, acid; specific gravity 1019; no albumin; no sugar; a few leucocytes and epithelial cells. The scars of the wounds in the feet were immediately opened, curetted out, and then swabbed with pure carbolic acid. Three grains of carbolic acid were ordered to be given hypodermically every three hours. The following day it was noted that the paroxysms were less frequent and less severe. Continuous drenching sweating was a marked symptom. Beginning at 2 A.M. the hypodermics were increased to five grains of carbolic acid every three hours.

July 25*th*. Patient decidedly more quiet and comfortable, though the opisthotonus is more marked. Examination of the urine—dark amber, clear, acid; specific gravity 1030; thin ring of albumin; no sugar, no excess of carbolic acid; a very few hyaline and granular casts present.

26*th*. Condition practically unchanged.

27*th*. Condition becoming worse; marked opisthotonus and risus sardonicus noted; pulse not so good; excessive sweating continues. Patient is unable to void urine spontaneously, but urine is perfectly clear. Carbolic acid increased to fifty grains daily.

28*th*. Slight improvement. Now voiding urine again; amber, clear, acid; specific gravity 1028. Faint trace of albumin; no sugar; one or two finely granular casts. No red corpuscles.

29*th*. Extremely sensitive to least disturbance. Carbolic acid increased to sixty grains daily. Urine still clear and freely voided.

30*th*. Temperature, which had been between 98° and 99° F. ever since admission, began rising yesterday, and at 4.30 this A.M. was 104° F. Paroxysms now almost continuous and patient rapidly weakening. Voided urine at 8.15 A.M.; light amber and perfectly clear; acid; specific gravity 1021; thin ring of albumin; no sugar; a few casts and a few red and white corpuscles. Chemically the urine showed a slight amount of carbolic acid. Patient became unconscious shortly afterward, and died at 10 A.M., temperature reaching 108° F. before and 110° F. after death. Autopsy refused. During a period of just one week the patient received a total of 303 grains of carbolic acid hypodermically without causing any smokiness of the urine at any time.

The report of the above case is of interest as showing the great tolerance of a patient with tetanus for carbolic acid, and the futility of the treatment in this case, though begun early and persisted in for one week. The case belonged to the class of acute cases most unfavorable

for treatment of any kind, and, although in this instance the treatment was begun promptly, its only effect seemed to be some lessening in the frequency and severity of the clonic spasms and possibly a slight prolongation of life.

MASTITIS COMPLICATING TYPHOID FEVER.

By Fellowes Davis, Jr., M.D., H. S. Patterson, M.D., and A. W. Hewlett, M.D.,
of New York Hospital.

Although the typhoid bacillus is recognized as the cause of many suppurative processes, mastitis of this origin is a very rare event. The authors are indebted to Dr. A. B. Ball for permission to report the following case:

Augusta A. (medical number, 5564), aged thirty-four years, janitress, was admitted to the New York Hospital on May 25, 1901, complaining of fever and weakness. She had never suffered a serious illness before.

Menses began when she was sixteen years old, and have been regular, scant, and painless. She has had two children, aged, respectively, six and three years, and one miscarriage five years before admission. Both children were nursed until they were a year old. The patient has never been troubled at any time with her breasts or nipples, and has never sustained an injury of the same.

She entered the hospital at the end of the third week of her fever, which had been without incident except for the severity of her bronchitis and the history of chills accompanied by vomiting. The latter were probably due to the administration of antipyretics.

On admission the patient's temperature was 105° F., respiration 28, and pulse 140 per minute. She was well developed and well nourished, but seemed listless. Lungs showed signs of a well-marked bronchitis. The spleen was not palpable. The abdomen showed a few typical rose spots. A positive Widal reaction was obtained on the day of admission. Ehrlich's diazo test gave a doubtful reaction. Blood cultures were made on May 27th, and again on May 31st (the twenty-third and twenty-seventh days of disease), but were negative for typhoid bacilli. Rose spots continued to appear for a week after admission, and two days later had entirely disappeared. Signs of bronchitis persisted until June 5th, the thirty-second day of the disease.

June 9th. Temperature touched normal on this the thirty-sixth day of the disease. (See chart.)

12*th.* Three days later the patient began to complain of pain in the left breast.

14*th.* The left breast is now swollen, and a hard and tender lump can be felt at its outer side The glands in the axilla are also enlarged and tender.

15*th.* The right breast also has now become tender. Lead and opium compresses were applied to both breasts, and for a time these seemed to improve. The patient, however, began to have a rise of temperature

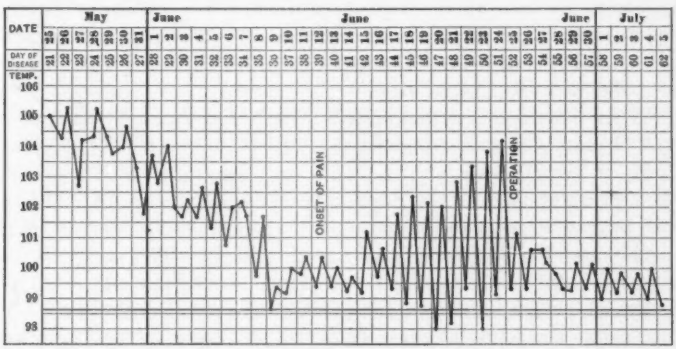
in the afternoon. (See chart.) This did not show the stepladder pattern of a relapse, but was of a septic type.

20*th*. The swelling and tenderness of the left breast has become much more marked, and involves the two upper and the outer lower quadrants. Skin freely movable; nipple not attached; no pus seen to exude from it after manipulation.

22*d*. Leucocytes, 13,200.

24*th*. Fluctuation was detected in the left breast. On aspiration a small amount of thick pus was obtained, from which cultures and cover-slips were made. The swelling in the right breast seems much diminished.

25*th*. The abscess in the left breast was opened, under chloroform, and about 150 c.c. of thick pus obtained. The abscess cavity occupied the upper and outer part of the breast, and consisted of many loculi, which were broken up with the finger.



Following the operation the temperature fell to normal, and healing was without incident. The painful lump in the right breast, which had been noticed first on June 15th, gradually diminished up to the time that the patient left the hospital, but even then a slight induration in the upper and outer quadrant could be made out. No exploratory puncture was made.

Bacteriological Examination of Pus Aspirated from Left Breast. Cover-slips show numerous pus-cells. A very few bacilli, but no cocci, were found. Cultures were made from the pus upon agar and in bouillon. The former gave a growth showing the characteristic features of the growth of the colon group upon agar. Smears showed a pure growth of a short bacillus. In twenty-four hours the bouillon showed a pure growth of feebly motile bacilli. On transplantation these gave in eight hours a growth of actively motile bacilli. This culture was tested with the blood-serum of a typhoid patient, and in a dilution of 1:10 gave a well-marked Widal reaction in ten minutes. Typhoid bacilli were present, therefore, in the abscess in pure culture.

The involvement of the breasts in typhoid fever must be a very rare event, if we may judge from the scarcity of the literature on the subject; yet it has been noticed several times by some observers. In 1874

Leudet[1] recorded five cases observed personally in which one or both breasts became tender and swollen, and in which inflammatory masses could be made out in the breast tissue. All resolved without suppuration. Vallin[2] reported a similar case. Curschmann[3] states that, in his experience, mild, transitory swelling of the breasts at the height of the fever is not uncommon, and may readily be overlooked, since the patient makes no complaint. He has seen similar swelling twice in young men. Suppuration is, he thinks, very rare. Fornaca[4] has recently reported a case of typhoid fever in which pain developed in the right breast on the twenty-ninth day of the disease, three days after the temperature had been normal. This was followed by a rise of temperature and by swelling of the breast, but no fluctuation could be detected. Five days later, on an exploratory aspiration, three drops of seropurulent fluid were obtained which showed typhoid bacilli on cultures. Later a similar trouble developed on the left breast, but aspiration was negative. As both breasts healed without abscess formation, this can hardly be classed as a suppurative case.

We have been able to find records of only four cases of suppurative mastitis associated with typhoid fever. In that reported by Charlotte P. West[5] the abscesses of the breast were associated with a general pyæmic condition. At the beginning of the third week of the fever both breasts became swollen and tender, which condition was associated possibly with the hypodermoclysis of salt solution. This condition disappeared. At the beginning of the fourth week, however, localized dusky red, swollen elevations and patches were discovered on both legs, both arms, and both breasts. Those on the right leg, left arm, and both breasts suppurated, and healed after free incision. The patient recovered. No examination of the pus was made.

In 1891 Destrée[6] reported two cases of typhoid fever in men in each of which an abscess developed in the right breast at the end of the third week. The pus from each abscess showed only staphylococci.

The fourth case, reported by Capellari,[7] almost exactly parallels our own. A girl, aged fifteen years, in the fourth week of an attack of typhoid fever, developed pain and swelling over the whole right breast. This resisted all palliative treatment. The temperature, which had touched normal, assumed the septic type. Fluctuation in the breast

[1] Clinique Médicale de l'Hotel Dieu de Rouen, Paris, 1874, pp. 96–99 and 113–118.

[2] Bull. de la Soc. Clin. de Paris, 1877, vol. i. p. 148.

[3] Der Unterlerbstyphus, Vienna, 1898, p. 184.

[4] Sulla mastite tifosa. La Riforma Med., 1900, vol iii. p. 266.

[5] Report of an Unusual Case of Typhoid Fever; Swelling of both Breasts, followed by Suppuration; Thrombophlebitis. Philadelphia Polyclinic, 1898, vol. vii. pp. 253–256.

[6] A propos de quelque cas de suppuration compliquant la fièbre typhoide. Journal de Médecine de Bruxelles, August 5, 1891.

[7] Ascesso mammario metastatico consecutivo a tifoide in adolescente. Corriere sanit, 1900, vol. xi. p. 88.

was detected, the abscess opened, and recovery followed. No bacteriological examination was made.

Mastitis accompanying typhoid fever, therefore, may or may not lead to suppuration. The latter condition is probably frequently overlooked, owing, as Curschmann suggests, to the apathetic state of the patient. The onset in Leudet's five cases was in the fourth week, and Curschmann speaks of its occurrence at the height of the disease. In the right breast of our patient it occurred in the sixth week. The favorable outcome in this class of cases led Leudet to the statement that inflammation of the breasts is a matter of no moment in typhoid fever, and heals spontaneously.

Suppurative mastitis in typhoid fever must be much rarer than the form which undergoes resolution. When not a part of a general pyæmic condition, it arises late in the disease, as do most of the other true typhoid suppurations. In Capellari's case and in our own the temperature had touched normal before the mastitis began. The course seems to be mild, and no difficulty was experienced after incision of the abscess. As with other abscesses, the fever was of the septic type, increase in the number of leucocytes was present, and the neighboring glands in the axilla were enlarged and tender.

Although both Curschmann and Capellari state their convictions that such abscesses are caused by the typhoid bacillus, Fornaca's case and the case here reported seem to be the first in which this causal relation is established. The source of infection is probably through the blood, for we know that in the majority of the cases of typhoid fever, typhoid bacilli can be obtained in cultures from the blood. We failed, however, in two attempts to do so in this case; but it must be remembered that these attempts were made late in the disease, when the chances of successful cultures are much diminished. In looking for a cause for a locus minoris resistentiæ in the breast, Capellari lays stress upon the age of the patient, believing that at puberty the mammary glands are less resistant, owing to their rapid growth. In our case it is worthy of note that the patient regarded the dry cupping, used on account of her bronchitis, as the source of her trouble. The breasts are, however, usually spared in cupping, which in this instance was mostly confined to the chest, behind, and the pain at cupping was more probably the effect than the cause of the early inflammation.

It has been shown that bacteria present in the blood may pass into the milk. Basch and Weleminsky,[1] however, believe that this is only possible where a lesion, especially a hemorrhagic lesion, is present in the mammary gland. In their experimental work with typhoid bacilli on guinea-pigs they failed to obtain typhoid bacilli in the milk, even

[1] Ueber die ausscheidung von Krankheitserregern durch die Milch. Jahrb f. Kinderheilkunde, 1898, vol. xlvii. p. 105.

when the bacilli were present in the blood in large numbers. Since we know that gross lesions of the breast may occur in typhoid fever, it seems probable that under such circumstances, at least, typhoid bacilli might appear in the milk of a nursing woman either during or after an attack of typhoid fever.

REPORT OF A CASE OF LYMPHOCYTOSIS, WITHOUT GLANDULAR ENLARGEMENT, COMPLICATED WITH PNEUMONIA.

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In presenting sections of the cerebral cortex of the following case, among a number of others of epileptic mania and status, to Professor Barker, of the Chicago University, the contents of the bloodvessels and the perivascular lymph spaces were so striking that the case seemed to merit a special study by itself. The condition was at first thought to have some relation to the gross changes found in the encephalon; but this was later disproved by the finding of the same condition in the vessels of the other organs. The patient was from the service of Dr. Stockton.

Mary C., white, single, aged thirty-eight years. She was an illegitimate child, and had spent most of her life in the county poor-house before being admitted to the hospital. She had been a patient in the hospital for more than twenty years, and nothing is known of her condition physically or mentally prior to this time. During her stay in the hospital she had frequent attacks of grand mal. At times the fits were very frequent, lasting for days, these periods being followed by periods of freedom from the attacks. For two years or more she had suffered from severe constipation, accompanied by cold and clammy skin, cold extremities, and increasing dementia. For the past year she had been very anæmic, and during the last two months of her life she became so weak as to be confined to her bed, and for the greater part of this time was the subject of a severe diarrhœa, with fetid, watery discharges. This condition was accompanied at times by nausea and vomiting, and abdominal tenderness was a constant feature. Her epileptic convulsions continued during this attack, and she was exceedingly irritable and quarrelsome. The skin was very sallow, and during the last week she had many fainting spells. Her pulse averaged from 100 to 120 to the minute, and her temperature ranged from 100° to 101° F. Death gradually followed from exhaustion.

The autopsy was held two hours after death, the organs being examined in the order in which they are given below.

The body is that of a well-nourished woman, length 151 cm., weight 78 pounds. Rigor mortis present in the inferior maxilla, not elsewhere. Livor mortis marked in dependent portions. Pupils equal and regular. Nasal and aural passages clear. Tongue dry and heavily coated.

The right submaxillary gland was enlarged, and on section emitted a large amount of pus. Smears from this pus contained diplococci, but were negative for tubercle bacilli. A number of purpuric spots were present over the trunk and extremities. Œdema of both lower extremities. Fingers were slightly clubbed. There was no glandular enlargement except in the right cervical region below the suppurated submaxillary gland.

Scalp, 4 mm. thick at the vertex, and very adherent to the calvarium over a depression 1 cm. in width at the upper part of the sagittal suture.

Circumference of skull	49.3 cms.
Diameters of skull—antero-posterior	15.9 "
biparietal	12.5 "
Thickness of frontal bone	1 cm.
" " temporal bone	4 mm.
" " occipital bone	1 cm.

There was no abnormality in the shape of the skull except the depression noted above, which, however, did not present on the inner surface. Pacchionian depressions were more marked on the left side than on the right.

The vessels of the dura mater were empty, not bulging. The dura was not abnormally adherent to the calvarium or to the soft membranes. Subdural fluid clear and not abnormal in amount. The inner surface of the dura and of both sides of the falx cerebri were covered with a thick layer of rusty, hemorrhagic deposit, which could be readily peeled away.

The convolutions on both sides of the encephalon, especially in the region of the Rolandic fissure, showed marked atrophy, and the arachnoidal fluid was correspondingly increased, filling the widened sulci and bulging the membranes outward. The fluid was clear and straw-colored. No granulations were found upon the ependyma of either lateral ventricle wall. The choroid plexuses and basal ganglia showed no macroscopical changes.

Weight of the encephalon and fluid	1 k.
" medulla, pons, and cerebellum	130 grms.
" left hemisphere	420 "
" right hemisphere	425 "
" fluid	25 "

The pons, cerebellum, and medulla showed no gross changes except a few minute granulations upon the floor of the fourth ventricle. Sinuses and dura at the base of the skull normal.

Sectio Corporis. The subcutaneous fat was lemon-yellow in color and measured 1.5 cm. at the umbilicus. The liver presented in the incision was very light yellow in color, and projected 1.5 cm. below the umbilicus in the right mammary line, or 13 cm. below the tip of the ensiform cartilage in the median line.

The muscles were pale and fairly well developed. The omentum projected well down into the pelvis. The stomach extended 13 cm. below the ensiform cartilage in the left parasternal line, *i. e.*, about 2 cm. in this region below the liver.

The diaphragm on the right side reached the third interspace; on the left side to the fourth rib. The apex of the heart lay in the fourth

interspace. The lungs and heart were displaced upward by the enlarged liver.

Left visceral pleura was densely adherent to the parietal layer by old, fibrous adhesions from the second to the fifth rib and over the posterior surface. Left lung weighed 360 grammes. Its interlobar pleuræ were densely adherent, and there was considerable hypostasis hæmatis in the dependent portions; otherwise the cut surface was crepitant and apparently normal.

Right pleural layers were bound together over their outer and posterior surfaces by numerous old adhesions. The lung was very similar to the left, except that in the posterior lower portion of the upper lobe there was a patch of pneumonia about the size of a lemon. This area was reddish-gray in color and non-crepitant.

The heart weighed 230 grammes. Thickness of left ventricle wall, 9 mm.; of right ventricle wall, 3 mm. The valves were normal, and the muscle very pale, but firm. The aorta had a number of sclerotic patches and calcareous plaques in its arch, and two calcareous plaques, 1 cm. in diameter, near the bifurcation of the abdominal aorta. Coronary arteries were normal.

The liver weighed 2340 grammes; 26 x 23 x 11 cm. Very large, light yellow in color; surface smooth; periphery of the lobules paler than the centre. The gall-bladder was filled with dark green bile, and contained no gallstones. The hepatic, cystic, and common ducts were normal and patent.

The pancreas and adrenals were normal.

The spleen weighed 240 grammes; 16 x 8.5 x 5.5 cm. Surface smooth, slightly mottled, and grayish in appearance. The substance was firm, cut with difficulty, and the cut surface was dark red in color, dry, and the Malpighian corpuscles plainly seen.

Ureters normal.

Left kidney weighed 130 grammes; 11.5 x 5 x 4 cm. The kidney was very pale, its surface was smooth except for a small retention cyst upon its posterior border; the capsule was more adherent than normal, and the cut surface was granular; width of parenchyma 2 cm., cortex 5 mm., medullary rays were wider than normal, and the glomeruli scarcely to be made out. The right kidney was very similar to the left.

The stomach was filled with a pale greenish mucous fluid. Its mucous surface was marked by longitudinal ridges, which were marked on their summit by red, hemorrhagic lines. These lines converged at a ring, 5 or 6 cm. from the pylorus, and then from here spread out again toward the pylorus. Measurements, 6 x 18 cm.

Uterus, vagina, ovaries, and Fallopian tubes normal.

Small intestine was normal except in its lower two feet. This portion was much congested, and on its mucous surface contained a number of small ulcers, irregular in shape, varying in size, with raised edges, necrotic bases, and injected peripheries.

The caput coli was intensely congested, and was studded over with small ulcers with gray bases, red peripheries, and raised edges. These ulcers varied in size from a pin-head to a bean. They extended from the caput to the rectum. In the rectal mucosa were several varicosities, and here and there a larger ulcer 1 to 2 cm. in diameter.

Larynx, trachea, pharynx, œsophagus, and thyroid gland normal.

Bacteriological examination negative, except that colon bacilli were obtained in the cultures taken from the kidney.

Microscopical Examination. The dura mater is thickened, and on its inner surface is a dense layer of connective-tissue cells in a state of active division. Inside of this, again, is a layer of hemorrhage in which the red blood-cells, intermixed with leucocytes, are undergoing degeneration, but whose outline may still for the most part be made out. Still further inward is another layer of connective-tissue corpuscles, and from both of these two layers of connective-tissue cells limiting the hemorrhage there is an extension into it, which in places has resulted in quite an extensive organization of the extravasation. Apparently the hemorrhage has taken place between the inner layers on the inner surface of the dura, raising the innermost layer with it. The vessels of the dura are the seat of a considerable thickening, affecting chiefly the intima and causing at times almost complete obliteration. In those vessels containing blood there is found a very large number of lymphocytes; in many places quite as many lymphocytes are found as red blood-corpuscles, and very seldom is it possible to find a polymorphonuclear cell among them. In the neighborhood of a number of the capillaries there are found minute hemorrhages into the lymphatic spaces, apparently recent from the good condition of the red cells and lymphocytes

The heart muscle takes the stain for the most part very poorly, and in places the striation is very indistinct. Gathered about the nucleus of the majority of the fibres, and most abundant in the palest of them, are minute granules of yellow pigment. In places also throughout the sections the fibres contain numerous minute fat-droplets. In the veins and capillaries throughout the heart muscle section is found the same increase in the number of small mononuclear cells.

The liver, owing to the very evident fatty condition, was injected with formalin and preserved entire in the same fluid, so that the portal system through which the fluid was injected was quite empty of blood. Where there was any blood remaining, however, there was found the same large number of lymphocytes as elsewhere. There was some commencing fibrosis evidenced, and in the portal areas the large increase in the number of lymphoid cells was very noticeable. The fatty change in the liver cells was extreme, and scarcely a cell was to be found which did not contain a large globule of fat pushing the nucleus to one side; the nuclei, however, still took the hæmatoxylin stain well.

The adrenal capsule presents nothing abnormal except that in the bloodvessels throughout its substance, and in the capsule, is found the same condition as has just been described.

In the pancreas, besides the increase in the number of lymphocytes in the vessels, there is some interlobular fibrosis.

The kidneys present well-advanced interstitial changes, especially involving the glomerular capsules; also thickening of the intima and media of the vessels. In the connective tissue throughout the organs are found areas of densely packed lymphoid cells. In all the vessels, in the capillaries of the tufts, and elsewhere, the great abundance of small mononuclear cells is very noticeable.

In the pneumonic portion of the lung a very strange and interesting condition is found. The alveoli are crowded with a variety of cells, including red blood-cells, large transitional cells, epithelial cells, large

phagocytic cells, lymphocytes, and polymorphonuclear leucocytes; but by far the vast majority of the cells are of the last-named variety, *i. e.*, the polymorphonuclear leucocytes. Still, when one turns to the blood-vessels and capillaries in the alveolar walls, there are very few polymorphonuclear cells to be found in their contents, but an abundance of lymphocytes is always found; especially is this so of the capillaries. Further, where one finds hemorrhages into the alveoli one finds always among the red blood-cells numerous lymphocytes, and these cells are almost wholly confined to these regions.

In the central portions of the ulcers the mucous membrane of the colon is quite destroyed, the floor of the ulcer being formed by the much thickened submucosa covered by a dense layer of pus-cells. The edges of the ulcer are shelving, and contain the remains of the tubules of the mucous membrane infiltrated with the lymphoid cells. The gland tubules further out near the periphery of the ulcers, and the submucosa underlying the whole ulcer, but especially its central portion, are very densely packed with lymphoid cells. There are also numerous lymphoid collections in the two muscle layers of the intestine in this region. The bloodvessels throughout these sections are filled with blood elements, but extremely noticeable is the very large number of small mononuclear cells present; here they form at times the majority of the cellular contents of the vessels, at times appearing almost as an embolus. The lymph follicles in the sections are swollen and densely packed with cells. The capillary vessels would seem often to end or open within the lymphoid masses.

In the stomach wall many of the papillæ have disappeared, the remains of the mucous layer are densely infiltrated with lymphoid cells, and on the surface are found many small hemorrhages. These were noted in the gross description as hemorrhagic dark lines. The same condition exists in the vessels here as elsewhere.

Sections were taken from the paracentral lobule, the precentral lobule, the temporal lobe, the medulla, and cord. These sections were stained by Nissl's method, thionin, eosin, and methylene blue and hæmatoxylin and eosin. In all of these sections the condition which first attracted the attention was very striking; that is, the presence within the vessels, and occasionally within the perivascular lymph spaces, of a great number of small mononuclear cells, in which scarcely any cell-body could be made out. These cells do not differ in any way from the small lymphocytes of the normal blood and lymphatic tissue, and are readily distinguished from the neuroglia nuclei, being much smaller, more deeply stained, and containing larger granules than the latter cell-nucleus. They are seen very readily in the Nissl and thionin preparations, and at times form almost an embolus in the smaller vessels. With the eosin and methylene stain no plasma cells could be found in the vessels.

The large motor cells in the paracentral lobule, in the medulla, and in the cord are, as a rule, normal; but among them one finds frequently cells in which the nucleus has an eccentric position, and occasionally one in which the nucleus is swollen and diffusely stained. In a number of them the nucleolus is found vacuolated; but this, as a rule, is quite regular in outline.

In the smaller and larger pyramidal cells of the cerebral cortex the nucleolus is almost always at the side of the nucleus, especially in the

former. The nucleolus is irregular in shape, with frequently spicules appearing at its circumference. The nucleus is at times swollen, filling almost the whole cell, and in these the nucleolus is pale and shrunken or has disappeared entirely. The dendrites are not seen for any great distance from the cell, but the chromatophile substance has in these and in the periphery of the cells often a very crumbly appearance. The Purkinje cells in the cerebellum are apparently quite normal.

It is to be regretted that the blood examinations were not made upon the above case during the life of the patient, and yet without doubt the case is one of lymphatic leucocythæmia, from the very great number of lymphocytes found in the bloodvessels. The value of the case is also somewhat lessened by the lack of a post-mortem examination of the bone-marrow, but as nothing in the case as reported suggested the condition found, and as this does not form part of the routine examination of the laboratory, it was neglected; through some mistake, also, the spleen was not placed along with the other organs for microscopical examination, still in spite of these deficiencies the case presents a number of features of interest which render it worthy of report.

These may perhaps be summarized before entering into any discussion of the case.

Clinically, constipation, followed by diarrhœa, pallor, rapid onset of terminal dementia, persisting epileptic attacks, fainting spells, and varying fever.

Post-mortem Examination. Suppuration of the submaxillary gland, purpura over the trunk and limbs, absence of lymph glandular enlargement, subdural hemorrhages, atrophy of the convolutions, extreme fatty liver, fatty heart, enlarged spleen, hemorrhages into the stomach and intestines, and chronic ulcerative colitis.

Microscopically: The presence of the very large number of lymphocytes in the bloodvessels, of all the organs, and the rarity of leucocytes of other forms, congestion of the veins, large increase in the lymphoid tissue in the alimentary tract and kidney, the apparent opening into these lymphoid masses of minute bloodvessels, the peculiar occurrence (*a*) in the pneumonic portion of the lung of polymorphonuclear leucocytes in the alveoli, with the persistence of the lymphocytes in the bloodvessels and capillaries, and (*b*) in the pus from the submaxillary gland and the changes in the cells of the cerebral cortex.

The case is further of interest in the support it would seem to lend to the theory of Ehrlich and Lazarus,[1] that in lymphatic leukæmia "the increase of the lymph cells is brought about by a passive inflow into the blood, and not by an active emigration from chemical stimuli."

At the present time the origin of the lymphocyte in the blood is generally accepted as the lymphoid tissue of the organism, since this cell conforms in every way to the lymphoid cell. Clinical evidence also would seem to prove a similar genesis for these cells in the bloodstream. In this case, then, with this origin granted and the lymph glands not enlarged, with the exception of a few in the neighborhood of the suppurating submaxillary gland, one is thrown for an explanation of their occurrence upon the spleen and lymphoid tissue elsewhere.

The spleen in the present case was larger than one usually finds at autopsy, and yet it comes within the limits of those given for normal weight of the organ (100 to 300 grammes[2]), weighing only 240 grammes.

The work of Kurloff, in Ehrlich's[3] laboratory, on the extirpation of the spleen in the guinea-pig has gone to prove that the spleen in this animal, at least, plays a very unimportant part in the formation of white blood-corpuscles. Kurloff found that the spleen was not indispensable, and that after splenectomy in the first year compensation occurs only in the lymph glands, followed in the second year by a great increase in the eosinophile cells. The study of cases of splenectomy in man, also, is analogous to the above, as the following conclusions, as a result of the researches by Hartman and Vasquez[4] on the extirpation of the spleen for tumors, will indicate:

1. A slight post-operative increase in the number of red blood-corpuscles and a true hyperleucocytosis occur and pass rapidly away.

2. Four to eight weeks afterward a lymphocytosis of varying duration is established.

3. Later, after many weeks, a moderate eosinophilia occurs.

It would seem from this that the exclusion of the spleen as a factor in the present instance is justifiable.

We have, then, left the great mass of lymphoid tissue in the intestinal tract and other organs, which, in this instance, is so much increased in amount; and, further, from the opening into these masses of lymphoid cells of small bloodvessels, which are abundantly filled with cells of quite the same type as those forming the masses themselves, it would seem very probable that their origin lies here, and that their presence in the vessels is a result of mechanical processes.

Ehrlich[5] has laid much stress upon the influence of the digestive tract in the production of lymphocytosis, the evidence coming mainly from the digestive leucocytoses, in which the lymphocytes are increased in number, from those cases of intestinal disease in infants in which lymphocytosis has been found,[6] and from typhoid fever, in the later stages of which a lymphocytosis is found.[7] No cases could be found in the literature at hand of differential counts made upon cases of chronic ulcerative colitis or of constipation, so that a comparison of the present case with others was not possible.

The question naturally arises, however, as to the priority of the two conditions found, viz., the chronic ulcerative colitis and the lymphocytosis. Was the condition of the bowel the primary one, with resulting lymphocytosis? or was there, as a result of some other toxin affecting the lymphoid tissue of the intestinal tract alone, a hyperplasia of this produced, which was later followed by ulceration, the original hyperplasia being responsible for the lymphocytosis?

From the lack of clinical data an answer to these questions is impossible. One might theorize without accomplishing, in the present instance, any result. The colitis was, however, of long standing, from the thickness of the bowel wall present and the extreme fatty condition

of the liver; also from the clinical evidence of prolonged constipation, followed by a diarrhœa of six weeks' duration, which ended in her death.

Of especial interest is the evidence in support of the theory that the lymphocytes in the blood-stream play no part in ordinary inflammatory processes. The case is allied to that of Neumann,[8] in which he produced suppuration in a case of lymphatic leukæmia, and in the pus found exclusively polymorphonuclear cells. Here, in the pneumonic portion of the lung, the alveoli are filled mainly with polymorphonuclear cells, and only where hemorrhage has occurred does one find lymphocytes within the air vesicles, although the capillaries in the alveolar walls contain lymphocytes in very large numbers.

In the pus of the suppurating submaxillary gland also the majority of the cells are of the polymorphonuclear variety, while in the vessels at the periphery the same majority of lymphocytes is found; still, the polymorphonuclear cells in the vessels are here not so rare as elsewhere.

The hemorrhages, subdural and into the mucous membrane of the stomach and bowel, the purpura, and the fainting spells are frequent concomitants of the condition of lymphatic leukæmia, and need no further comment here.

The changes in the nerve cells of the cortex were probably due in part to œdema, as described by Hoch,[9] from the lingering nature of the death of the patient, in part to the toxic condition, and possibly also in part associated with the macroscopic atrophy of the convolutions.

While the incomplete nature of the above report renders it less valuable than could be desired, yet the following conclusions would seem to be justified:

1. That a lymphocytosis may result, where there is absence of enlarged lymph glands, from increased lymphoid tissue in the intestine.

2. That the presence of these cells in the blood-stream is probably due to mechanical causes (Ehrlich).

3. That the lymphocytes in the blood-stream play no part in ordinary inflammatory conditions.

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SYMPTOMS POINTING TO THE NECESSITY FOR OPERATIVE INTERFERENCE IN MASTOID SUPPURATION.[1]

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The diagnosis of suppuration of the mastoid antrum and cells is a subject of much interest to the otologist.

In the majority of cases it is not difficult for one who has had considerable experience to make such a diagnosis. To the less experienced it is a more formidable undertaking; while to those having no special knowledge of ear diseases the diagnosis is never made until the symptoms have become so apparent that, almost, "he who runs may read" them.

The great importance of diagnosis in this diseased condition cannot be overestimated, and he who makes an early diagnosis renders valuable service to his patient.

In selecting the proper time for operative interference in mastoid complications of suppurative middle-ear disease the judgment of the operator is frequently subjected to a severe test. While opinions may vary to a marked degree, from operating at the very commencement of mastoid involvement to a delay which may lead to extensive complications, there certainly must be a period in the history of every case when operation should be no longer delayed, and the object of this paper is to endeavor to outline those symptoms which may be looked upon as pointing to the necessity for operative interference. With opinions and experiences so varied, as no doubt have been those of the otologists present at this meeting, it is not to be expected that the conclusions of anyone will meet with the approval of all. For convenience the topic will be divided into chronic and acute mastoid suppuration.

By acute suppurative involvement of the mastoid we mean an extension of the suppurative process from the middle ear into the mastoid antrum and cells during an acute attack of middle-ear suppuration. This is the generally accepted definition. On the other hand, during a chronic middle-ear suppuration there may occur an acute mastoid involvement. This last-named condition is, however, more often spoken of as chronic.

A discussion of the indications for operation upon the mastoid in acute cases is specially fitting at this time, when—partially as a result of the season through which we have passed, but more so as a result of the prevalence of the grip—all practitioners of medicine are being called

[1] Read at the Eastern Section meeting of the American Laryngological, Rhinological, and Otological Society, held at Buffalo, N. Y., 1901.

upon to treat numerous cases of acute suppuration of the middle ear. In all grip cases there is present a large amount of infectious material in the nose and nasopharynx. The characteristic cocci of this infection are the streptococci, staphylococci, and pneumococci. The accompanying inflammation and tumefaction of all the intranasal tissues so obstruct the nasal channels that ordinary blowing of the nose is attended with more or less danger, for some of the infection may enter the Eustachian tube and become the exciting cause of acute middle-ear suppuration—a suppuration which is peculiarly violent, virulent, and rapidly destructive to all tissue with which it comes in contact.

It would seem that physicians in general do not fully comprehend the very great danger to the ear attending attacks of the grip, and it is equally true that much of this danger could be avoided by proper treatment of the nose and nasopharynx during the time of its ravages. To keep the nose and nasopharynx free from infection, or comparatively so, is to render the Eustachian tube much less liable to an invasion of bacilli.

It should be the routine practice in both private and hospital practice to make microscopical examinations of the pus in all acute cases of middle-ear suppuration, and if streptococci or staphylococci are present we know the tendency to mastoid involvement to be much greater than otherwise would obtain. When microscopical examination of the pus is made the report should state which cocci predominate. It has been rather conclusively shown that pus is more virulent when the streptococci are in excess.

Blood examinations also, while not absolutely necessary in uncomplicated cases, may be of greater service when infection of the lateral sinus is expected, and it is strongly urged that in all cases where general infection is suspected such examination should be made.

Other things being equal, knowing the rapidly destructive nature of the pus just described, with its tendency to migrate into tissues far beyond the confines of the middle ear, even to the blood sinuses and brain tissue, the knowledge of this pus characteristic would lead one to operate much earlier than if it appeared upon microscopical examination to be free from this infection. Acute mastoid disease may be described as mastoid involvement appearing during the course of an acute suppuration of the middle ear, the symptoms of which may be described somewhat as follows: Always bear in mind that upon these symptoms must be based one's final judgment as to operation. Perhaps the most marked symptom of mastoid involvement is pain. This pain usually comes on some time after the excruciating pain which precedes suppuration of the middle ear has passed away and after the discharge from the ruptured or incised drum has been established. It is a dull, heavy pain, not definitely localized like the pain of acute suppuration of the middle ear, but rather

diffused over the surface of the temporal bone. Patients frequently describe a feeling like a weight upon the head. This pain is not so severe as in the middle ear, and is located behind and above. With it the patient describes a feeling of fulness, heaviness, and pressure over the entire parietal region. The pain may or may not be constant, and may or may not depend upon free or diminished discharge from the external canal, but it is usually present at least to a slight degree. Another prominent symptom is tenderness upon pressure. This tenderness is usually marked at the tip of the mastoid, but is more significant when it is present higher up, just over the mastoid antrum; and more recently we have come to attach more importance to tenderness in this region than to the same condition when lower down at the tip. The mastoid tip may be tender on pressure when no disease is present. Temperature must also be considered, but it must not be relied upon as pathognomonic in any degree in mastoid involvement. In young children it is usually present and rather high. In adults, even with extensive mastoid involvement, the temperature may not be particularly disturbed. It must be borne in mind, however, that in certain complications like sinus infection the temperature may be very high or subnormal, in which case it becomes a very important factor.

A not unimportant symptom of mastoid suppuration is the facial expression. This expression is not especially one of agonizing pain; the countenance rather is indicative of anxiety; it is altogether an extremely unhappy expression. The head usually hangs forward, with an inclination to lean toward the healthy side. To one whose privilege it is to see large numbers of mastoid cases the observation of this symptom must become a valuable adjunct in completing the diagnosis. I have frequently, from a row of waiting patients in the clinic-room, pointed out cases suffering with mastoid complication from the facial expression alone.

External periostitis, with or without infiltration of the soft tissues, certainly must be considered as a symptom, although, as a rule, operative interference should be resorted to long before these symptoms appear. When present they must be considered rather as a complication than as a symptom of the disease itself. The drum membrane must not be overlooked in making a diagnosis of mastoid involvement, especially that portion known as Shrapnell's membrane or the attic region. When this membrane, together with the posterior superior portion of the wall of the canal, is found to be bulging downward and forward into the canal, giving it the appearance of being narrowed from above downward, a picture is given which constitutes one of the two most prominent symptoms of mastoid suppuration. The other prominent symptom to which I refer is tenderness upon pressure over the region of the antrum. To define the relation of these two symp-

toms to surgical interference, I would say that prolonged tenderness upon pressure over the region of the antrum, together with bulging of the atticus tympanicus and the superior posterior wall of the canal, constitutes sufficient reason for operative procedure, and especially so when the pus contains streptococci or staphylococci.

Suppuration in the mastoid bone must be considered as a complication of suppuration of the middle ear; but there are many further complications which may and often do complicate the mastoid suppuration. The symptoms of these complications, while not strictly a part of this paper, the scope of which is confined to the indications for operating, may, however, be briefly mentioned. I refer to rigors, vomiting, vertigo, choked disk, aphasia, paralysis of the extremities, high temperatures, facial paralysis, dull mentality, uneven pupils, so-called typhoid conditions, etc. All of these symptoms indicate that the suppurative process has gone far beyond the mastoid cells into either the lateral sinus, the dura, the cerebrum, or the cerebellum, and when these symptoms are present we know that there can no longer be any possible doubt as to the necessity for operating. It may be possible to have a non-suppurative involvement of the mastoid cells, a mere congestion; but such a condition is hardly probable. The first stage of a suppuration may be a catarrhal congestion, but we must be led to conclude that when inflammation of the mastoid cells is present that involvement is suppurative in character. This opinion is greatly strengthened by the fact that we practically find mastoid involvement only in the presence of middle-ear suppuration. Knowing this to be true, the moment the symptoms of mastoid suppuration become apparent we are face to face with the necessity for a decision whether or not we shall operate. The very early symptoms may be and sometimes are relieved by measures other than external operation. Free incision of the drum membrane carried well through the attic region into the external canal, allowing the escape of pus, will often completely relieve all indications of mastoid involvement. In fact, the whole theory of such relief is based upon the importance of free drainage, so that even when the antrum itself is involved the necessity for further involvement of the mastoid cells is taken away by establishing such drainage. This, together with local blood-letting by means of real or artificial leeches, and with the ice-coil or hot poultices, should be abortive in a small percentage of cases. During the early stages of middle-ear suppuration, and even after mastoid symptoms appear, irrigation with hot sterile water is commendable treatment.

Hot douches, when used, should be from 100° to 110° F., and large quantities of water should be employed at each treatment. These treatments should be frequent, the douche being used once or twice each hour.

The great tendency is to make use of these for too long a time; I refer especially to the ice-coil and to the hot poultice. The ice-coil

should not be used longer than twenty-four or thirty-six hours, after which time it may become a dangerous application on account of its proneness to mask the symptoms. Continued use of poultices may be almost as bad; in fact, any prolonged attempts to abort mastoid suppuration are to be deprecated. In my judgment the greatest results in the way of prevention are to be obtained from free drainage of pus through the drum, although the other methods should be given a fair trial, providing they are used early in the history of the case.

At the very onset of mastoid involvement the patient should be placed in bed, and he should remain there until all symptoms have passed away; in short, he should be treated as any other patient suffering from a serious disease.

Next to free draining, this procedure should be given the highest place in the list of preventive measures.

External operation should be performed in acute suppurative inflammation of the mastoid cells when a permanent remission of symptoms has not been brought about by free drainage through the drum membrane, or by the application of ice-coils or poultices, or from local bloodletting, and when this time has arrived there should be absolutely no delay in operating—the sooner it is done the better it will be for the patient. The majority of the hospital cases and many of the private cases have already reached this stage before we see them, and it is a mistake to attempt to abort under such circumstances. It is at this very point that many otologists waver, and, by still further delay, complications arise which may make the operation much more extensive and jeopardize the patient's life. Just when the exact time arrives may not be measured by days or hours. The date must be determined by the good judgment of those who have the patient in charge.

Many delays in operating come either from lack of willingness to consent upon the part of the patient or his family or from the extreme conservatism of the family physician. Conservatism is a quality not to be ignored, but when carried to the extreme it reacts to the great detriment of the patient. The same statement may be equally true when applied to radicalism. Indeed, it is quite possible for the conservative to be radical in his conservatism, and of the two it would seem to be preferable that one should be conservative in his radicalism rather than radical in his conservatism.

Another strong point in favor of early operation is the better hearing results to be thus obtained. Early operation usually will be followed by practically perfect hearing on the affected side.

It must not be forgotten that pus pent up in the mastoid cells is in a position seriously to menace nearby vital structures. Not more important is it to remove the suppurating appendix than to open suppurating mastoid cells.

In my experience, especially in grip cases, the destructive process is so rapid that one frequently has to operate soon after the onslaught of the disease.

A recent experience may serve to indicate what is meant by the above statement.

CASE I.—"X.," child, aged eight years, had an attack of grip commencing January 10, 1901. January 12th pain in the ear began, followed by suppuration. On the 14th pain was relieved until the 16th, when severe mastoid symptoms manifested themselves; by the 19th the mastoid had become very tender, and on the 20th, when I first saw him, the symptoms were so severe that the question of palliation was not to be considered. Operation on the 20th revealed pus not only in the antrum, but in the cells at the very tip of the mastoid, so that three days after the first mastoid symptom the proper date for operating had come. In fact, it would have been better in this case could the operation have been performed a day later.

CASE II.—W. W. W., male, aged thirty-two years, had an attack of grip dating from April 1, 1901. During the night of April 4th he had a severe attack of middle-ear pain; April 5th I first saw him and found the drum membrane bulging both in the attic and posterior quadrant. A free incision was made, which was followed by pus and free hemorrhage. On April 6th mastoid tenderness developed; on April 7th he was admitted to the hospital, where the hot douches were continued. He remained in bed during the entire illness. On the 9th, at noon, the mastoid was opened and excessive involvement was found, the bone being softened and necrotic over a wide area, and the lateral sinus was fully exposed during the operation; the pus in this case was found to be loaded with streptococci, staphylococci, and pneumococci. These two cases illustrate the rapidity with which the mastoid may become involved in certain cases.

If microscopical examination of the pus from the middle ear in a case presenting symptoms of mastoid involvement reveals the presence of streptococci, staphylococci, and pneumococci, it is my belief that no amount of palliative treatment will have any effect upon the final result, and we usually find this condition in grip cases, so that it would seem wise, especially in this class of cases, not to delay operative interference; this is especially true if streptococci predominate. I do not mean to be understood as advocating an operation during the first few hours of mastoid involvement in the cases referred to above, because during that time palliative measures should be resorted to. It is agreed by many operators that even if pus be not found no harm has come to the patient from operation, as a patient always recovers; but to me this argument does not appeal. It is unfair to subject a patient to an operation simply because it will do him no harm. In mastoid operations it is necessary that we positively determine mastoid involvement, after which there should be no hesitancy as to the necessity of operative interference. It is possible that to delay operative interfer-

ence may not result in great danger to the patient, and occasionally an apparent temporary recession may be obtained, but even under these circumstances the whole trouble may light up anew at any time, making operation necessary, and delay of this kind must be attended with more or less danger of still further complications, many of which are attended with serious menace to life.

With such views as have been expressed in this paper in favor of early operative interference one might expect that in a large hospital service a fair percentage of the cases operated upon would reveal no pus or diseased tissue; but so far as my own experience is concerned this is not true; neither is it, I believe, in the service of others in the large hospitals.

During the last season I have not failed to find pus or necrosed tissue in a single instance.

The method of operation adopted should carry out this one prime necessity, namely, that all diseased tissue should be removed. The location of the incision, its length, and the method of removal of the external table may vary, but these variations are not of very great importance so long as the vital point above mentioned is carried out.

It is not the purpose of this paper to enter far into the domain of treatment, but it has seemed wise to call attention to the necessity for thoroughness.

A word or two in regard to the Wilde incision: Aside from such benefits as come from local blood-letting, nothing may be expected from this incision except the annoyance of having an open wound to contend with in a nearby location. To me the Wilde incision has always seemed a very wild incision to make. It has been stated that if upon making the Wilde incision the external table seems healthy no attempt should be made to enter the cells. Experience, however, does not bear this out, as patients suffering from mastoid involvement should be given the benefit of operative relief long before the disease has progressed far enough to break through the external table.

Local blood-letting may be obtained by much simpler methods, and there can be no necessity for ever making a Wilde incision.

Chronic Suppuration. Long delayed suppuration of the middle ear attended with involvement of the attic, and possibly the antrum and mastoid cells, may lead to the necessity for external operation in order to overcome the suppurative process. This is especially true in cases of chronic otorrhœa, the discharge of which is offensive, and with the presence of cholesteatomatous masses and carious bone. Only by external operation in many instances are we able to indulge in any hope of permanent relief. That a chronic otorrhœa is attended with more or less danger to life will not be denied. Such danger may not be great, and fatal cases may not be frequent, but the danger still exists. It is

well known to most of you that life insurance companies at the present time refuse absolutely to accept as risks any persons who suffer from chronic discharge of the middle ear. An attempt should certainly be made to cure permanently all cases of chronic otorrhœa. Local measures—removal of the necrosed ossicles and of other known methods—should be made use of before resorting to external operation.

INFECTION OF THE RECTUM, WITH SECONDARY INFECTION OF THE LIVER, CAUSED BY THE BACILLUS INFLUENZÆ SIMILIS.[1]

By W. Ophüls, M.D.

(*From the Pathological Laboratory of Cooper Medical College, San Francisco, Cal.*)

The following case of what appears to be an infection with a bacterium belonging to the group of the influenza bacillus seems interesting enough to be put on record. I am greatly indebted to Dr. J. O. Hirschfelder for his allowing me to make use of the clinical history of the case in this paper.

J. B., aged thirty-six years, single, Austrian, laborer in mine, admitted November 29, 1900. Service of Dr. J. O. Hirschfelder. On admission: temperature 102°, pulse 100, respiration 48. Patient complains of severe chills, cold on chest, cough, and general malaise. No diseases of childhood. Father died of smallpox, mother of unknown cause; one brother alive and well; denies venereal history; drinks moderately and smokes excessively; has never been sick before, except with severe cold.

Present trouble came on eight days ago. On coming from Angel's Camp to Stockton he took a severe chill; was compelled to lay off for four or five days in Stockton. Then came to San Francisco; was in bed five days, while in Stockton most of the time. Arriving in San Francisco he went to the Receiving Hospital, and remained overnight. Was brought to the City and County Hospital November 28, 1900. Complains now (November 29th) of chills, followed by sweats, cough, expectoration of blood and mucus, sore-throat, headache, loss of appetite, diarrhœa for the last week, and jaundice.

Physical Examination (Dr. Hirschfelder). Patient well developed, fat, markedly icteric; head, no peculiarity; pupils equal, react to light; no scar in pharynx; gums normal; neck, nothing abnormal; cervical glands slightly enlarged; thorax well developed, deep, broad. Right side moves less than left. Tongue slightly coated. Flatness of right lower lobe. Bronchial respiration and râles of varying size. Sputum brownish-red, mucous. Area of cardiac dulness: third rib, left sternal border, fifth space, 2 cm. left of mammillary line. Tones clear, marked accentuation of second pulmonic sound. Abdomen distended; region of stomach sensitive; no tumor palpable; liver, 13 cm. in diameter;

[1] Read at the Thirty-first Annual Session of the Medical Society of the State of California.

consistency somewhat increased. In region of gall-bladder smooth, round mass felt, not sensitive. Spleen extends to anterior axillary line, not palpable. Right inguinal hernia, easily replaced. Inguinal glands slightly enlarged. No scars on penis.

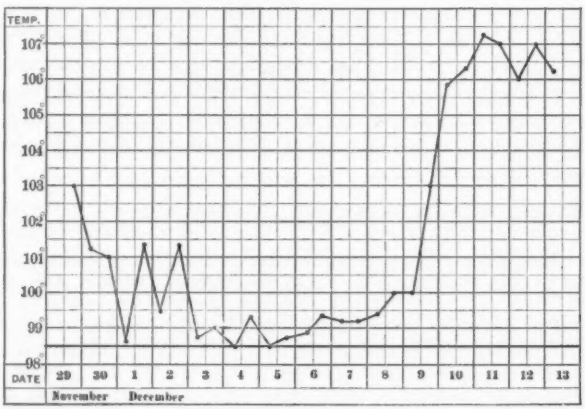
November 29th. Patient decidedly icteric; had severe nose bleeding. Pulse 82, full, regular.

30*th.* Nose bled twice during the day.

December 1st. Nose bled twice this morning; sweat some during the night; tongue coated with brownish-gray coating. Dulness in right lower lobe of lung continues; liver, 11½ cm. in diameter; abdomen somewhat distended. Region of gall-bladder sensitive; spleen not enlarged.

3*d.* Urine: specific gravity 1016; reaction acid; albumin, trace; sugar, diazo reaction, indican negative. Bile pigment present.

5*th.* Patient decidedly more icteric.



10*th.* Patient somewhat more icteric. Pulse 80, full. No dulness of the lungs, vesicular respiratory murmur. Area of cardiac dulness: lower border of third rib, left sternal border, sixth interspace, 2 cm. to left of mammillary line; tones clear; liver 9 cm. in diameter, consistency somewhat increased; gall-bladder not palpable; spleen not enlarged; abdomen not distended.

11*th.* Pulse 106, respiration 46. Tongue coated with grayish fur; right pupil somewhat larger than left; no dulness of lungs; both tonsils somewhat reddened; on right tonsil yellowish deposits.

12*th.* Pulse 108, respiration 48. Slight tonsillitis; very small patch on right tonsil; lungs normal; numerous râles are heard over anterior portion of chest; systolic murmur at apex and pulmonic valves; tones at apex indistinct; liver 10 cm. in diameter; spleen not enlarged; abdomen somewhat distended with gas; region of gall-bladder sensitive; gall-bladder palpable; slight ileocæcal gurgle; tongue moist, coated.

13*th.* Patient decidedly more icteric. Pulse 116, fairly strong; tongue coated; dulness of right lower lobe of lungs; bronchial breathing; a few râles; liver, 16½ cm. in mammillary line; to the right of

median line, 13½ cm.; consistency increased; markedly sensitive; gall-bladder palpable, painful; spleen enlarged, not palpable. Patient is so dyspnœic that character of cardiac tones cannot be determined.

Blood: White corpuscles, 7200 in c.mm.; polymorphonuclear leucocytes, 80 per cent.; small mononuclear leucocytes, 14 per cent.; large mononuclear leucocytes, 5½ per cent.; eosinophiles, ½ per cent.

Post-mortem Record. J. B., aged thirty-six years, laborer, died December 14, 1900, 8 A.M.

Autopsy, December 14th, 10.30 A.M. Strongly built, muscular, moderately well-nourished man. Shoulders slightly stooping. Slight enlargement of the femoral lymphatic glands. Slight cyanosis of lower face and neck. Skin and mucous membranes are bright yellow. Abdomen a little distended; marked rigor mortis. Liver projects two finger-breadths below free margin of ribs in mammillary line. The lower end of the omentum is fastened to the hernial sac of a small inguinal hernia on the right side. Lower border of stomach at umbilicus. A few drops of clear, yellow fluid in rectovesical pouch. Diaphragm at the fourth rib on both sides. Thorax broad, symmetric. Stringy, easily broken adhesions in the lower part of left pleura. The larger part of the right pleural sac obliterated by similar adhesions. About 100 cm. of clear, dark, yellow fluid in pericardium. Both sides of heart distended and filled with dark fluid blood. Right ventricle averages 3, left 12 mm. in thickness. Valves, aorta, coronary arteries normal, except for bright-yellow discoloration of intima and endocardium. Heart muscle normal.

Left pleura, many small hemorrhages in adhesions. Bronchi on left side empty, cyanotic. Lower lobe and posterior part of upper lobe of left lung contain little air and much blood. Anterior portion of lung normal. Right lower lobe firmly attached to the upper surface of the diaphragm. Numerous small hemorrhages in adhesions. About in middle of lateral surface of upper right lobe there is a bright yellow, dull spot in pleura, measuring about 2 cm. in diameter, and surrounded by a hyperæmic zone. The pleura in the neighborhood is a little opaque and rough on surface. Beneath the discolored pleura there is a necrotic area of light-yellow color extending about 1 cm. into the pulmonary tissue. Around it is a hyperæmic zone. The bronchial tubes on the right side contain a little bloody mucus. In one of the large bronchi of the lower lobe one finds enveloped, in a little bloody mucus, a hard, stony, irregular, grayish-white, non-obstructing concretion, 7 x 5 x 3 mm. in size (quite apparently a concretion from the tonsils or the ducts of one of the salivary glands that was aspirated shortly before death).

The mucous membrane of the bronchi on the right side is anæmic, intensely icteric, otherwise normal, even in neighborhood of foreign body. The right lower lobe of lung contains little air. Rest of lung normal. Spleen, 15½ x 8½ x 4½ cm. A few just visible white thickenings of capsule on convexity. The organ is hard, cut surface smooth, dark red. Markings distinct. Adrenals normal. Left kidney: capsule a little adherent; size, 14½ x 6½ x 3 cm. Consistency soft. A considerable amount of fatty tissue around renal pelvis. Cortex appears a little cloudy. Right kidney same as left. Appendix normal. Right testicle: slight thickening of the outer layer of the tunica vaginalis. Testis itself normal. Bladder contracted, contains a few drops of dark-

yellow urine. Prostate a little large. Seminal vesicles normal. Rectum empty. Just above anus the mucous membrane shows some thick hyperæmic longitudinal folds, which are partly covered with brown sloughs. On incision two small abscess cavities are found in submucosa, near the sloughs on surface. Pelvic veins contain fluid venous blood, no macroscopic thrombi. Contents of duodenum strongly acid, not bile-stained. There is a good deal of mucus in them. Similar contents in stomach. On pressure a little orange-colored bile is discharged from bile duct. The ducts themselves are not obstructed, and are of normal size and appearance. Gall-bladder small; wall œdematous. Portal vein contains a little dark fluid. Hepatic artery normal. Stomach greatly distended. Mucous membrane in large folds, swollen, cloudy. Pancreas normal. Vena cava inferior, normal. Liver: surface shows a large number of bright yellow spots of size of millet-seed, most of them arranged in groups of twenty or more. They are surrounded by hyperæmic zones. Similar areas are found scattered all over cut surface. The small branches of the hepatic vein in their surroundings contain soft yellowish-gray thrombi. In the comparatively normal parts of the liver the lobuli show dark greenish-brown centres and light yellowish-brown peripheries. No thrombi in branches of portal vein. Slight opacity of the peritoneal covering of the right lobe of the liver. Jejunum distended, mucous membrane swollen, cloudy. Ileum, colon, appendix normal, contents moderately bile-stained. Sigmoid flexure shows slight irregular hyperæmic swellings of mucous membrane. It is covered with thick, viscid mucus. Slight enlargement of the thyroid. Rest of the organs of neck normal.

Diagnosis. Proctitis, with sloughing and abscess formation in submucosa; multiple metastatic abscesses of liver; septic thrombosis in hepatic veins, septic infarction of lung; gastro-enteritis; green icterus of liver; general icterus; pleuritic adhesions on both sides with hemorrhages in them; slight chronic and acute parenchymatous nephritis; enlargement of the thyroid; inflammatory enlargement of the spleen.

The result of the microscopical examination is the following:

Rectum. (Figs. 1 and 2.) In a number of places the mucous membrane and also part of the submucosa are destroyed. These small but deep defects are filled in and covered with old blood clots. The defects are surrounded by a thin layer of necrotic tissue, then follow layers of tissue which are very hyperæmic, full of small hemorrhages, and moderately infiltrated with lymphocytes and polymorphonuclear leucocytes. The connective tissue cells in these areas are large and seem to be increased in number. Many of them are full of minute fat droplets. There also seems to be some increase in the connective tissue fibrils.

The larger portion of the mucous membrane has normal structure and is covered with a thick layer of mucus. Many minute bacteria in irregular heaps in lower parts of blood clot and in tissue around defect. In Fig. 2 there is a small vein at the edge of which there are two large heaps of these bacteria.

Liver. (See Figs. 3 and 4.) The centres of the liver lobules show some stagnation hyperæmia, slight thickening of central veins, icteric pigmentation and stagnation of bile in bile capillaries. Many of the liver cells in centres of lobules are small and full of fat droplets. There are very many moderately large irregular areas in which the hepatic tissue is completely necrotic. In the necrotic material and in the liver cells

FIG. 1.

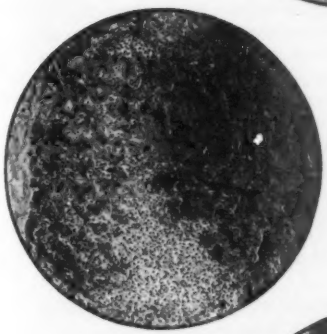


FIG. 3.

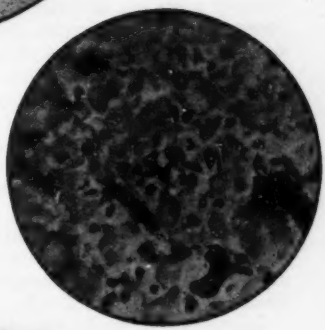


FIG. 4.

FIG. 2.

in surroundings of necroses there are some minute fat droplets. The necrotic material appears finely granular under the microscope. In the middle of some of the necrotic areas there is evidence of beginning liquefaction. The necrotic layers are surrounded by a cellular layer which consists largely of epithelioid cells with some lymphocytes and polymorphonuclear leucocytes. The cells near the necrotic material show pyknosis, caryorrhexis of their nuclei, and other signs of degeneration. Small branches of the hepatic vein in the neighborhood of these areas show fatty degeneration and necrosis of their wall. They are filled with thrombi that consist of fibrin, agglutinated blood platelets, and small heaps of fat granules. In the periphery of the necrotic areas and in the outer layers of the cellular tissue around them there are very many minute bacteria in thick, irregular heaps which in many places are situated in and follow the course of the capillaries. Similar bacteria are found in thrombi of the hepatic veins. No cocci to be found in sections stained with Gram's method. The bile ducts, unless secondarily involved, are normal.

Lung. Right upper lobe. Large, irregular necrosis. At apex of necrosis a branch of pulmonary artery, which is filled with a thrombus consisting of agglutinated blood platelets, leucocytes, and some epithelioid cells. In periphery of necrosis one finds the tissue infiltrated with the same cells as those which surround the necroses of the liver. The polymorphonuclear leucocytes are perhaps a little more numerous. The pleura over necrosis is partly necrotic, in other places hyperæmic, contains some small hemorrhages. It is covered with a thin layer of fibrin. Between fibrin and pleura in many places a row of swollen endothelial cells is plainly visible. Many minute bacteria in irregular heaps in periphery of necrosis and in embolus in branch of pulmonary artery. No cocci in sections stained with Weigert's modification of Gram's method. The comparatively normal parts of lung show marked thickening of septa, some œdema, a few hemorrhages, proliferation and desquamation of epithelial cells.

Jejunum: Lymphocytic infiltration of mucous membrane, many goblet cells in epithelium, much mucus on surface.

Stomach: Much mucus on surface, no structural changes.

Spleen: Hyaline degeneration of some of the arteries, hyperæmia, fatty degeneration of some of the cells of pulp.

Kidney: A few scattered small areas of chronic nephritis, icteric pigmentation, slight fatty degeneration in spots.

Thyroid: Follicles large, much colloid.

Tonsil: Fatty degeneration of germinal areas of follicles.

Testis: Normal.

Aorta: Slight irregular thickening of intima, with very marked fatty degeneration of connective tissue cells.

Result of Bacteriological Examination. Cover-slips from abscesses in submucosa of rectum, necrosis of liver and lung show very many minute (largest diameter about 0.85μ) bacteria. They are mostly rod-shaped, but some of them are round, coccus-like. They stain hardly at all with methylene-blue, a little better, often somewhat irregularly, with carbol-fuchsin. Some show polar staining. Some appear as diplobacilli, or, again, as diplococci. In spots they form small clusters; here and there one finds a short thread. They do not stain with Gram's method.

Cultures taken at post-mortem (smear cultures on glycerin agar-agar).

Liver necrosis: two colonies of colon bacilli. Three minute dewdrop-like colonies of minute bacilli. One of the colonies is contaminated with streptococci.

Pus: Abscesses in submucosa of rectum; many colonies of colon bacilli; a few minute colonies like those in culture from necrosis of liver.

Contents of gall-bladder: Two colonies of colon bacilli. Three minute colonies like those in culture from necrosis of liver.

Lung, right lower lobe: Two colonies of staphylococcus pyogenes aureus; one colony of colon bacteria.

Spleen: No growth.

Kidney: One colony of staphylococci; two just visible colonies of streptococci.

The small bacilli that are found so abundantly in the sections and were isolated from necrosis of the liver and suppurating areas of rectum show the following cultural peculiarities:

Glycerin (6 per cent.) serum; no growth. Löffler's serum at 37° C.; after twenty-four hours just visible dewdrop-like colonies appear, which in two or three days grow to about 2 mm. in diameter and become a little more opaque. The centre of the colonies is a little raised and the margin slightly irregular.

Glycerin agar-agar, 37° C.; after twenty-four hours just visible dewdrop-like colonies appear, which later enlarge a little but not very much. They always remain transparent, have slightly irregular margin, raised centre. Macroscopically, and also under the low power of the microscope, they cannot be differentiated from colonies of pneumococci or a delicate growth of streptococci.

Sugar agar-agar; slight granular growth along track of needle; no fermentation.

Beef-tea remains clear; after a few days a little, finely granular sediment appears.

Milk[1] slowly acidified, not coagulated.

Potato: No growth.

Gelatin: No visible growth even after several weeks.

The growth on solid media is quite characteristic in that it forms a delicate grayish-white film that hangs together very tenaciously, so that in trying to remove some of it with the platinum needle, usually the whole colony or larger pieces of it come off. It is also impossible to obtain a good emulsion of them in water. The film, on being rubbed over the cover-slip, breaks up into small fragments, but one never achieves a separation of the individual germs. The just visible grayish-white flakes in the beef-tea are large clusters of bacteria, and show the same peculiarity. The bacteria are non-motile. They have about the same size as those found in the tissues, being, perhaps, slightly larger (about 1μ long). They stain badly with methylene-blue and even with carbol-thionin, well with dilute (1 : 5) carbol-fuchsin. For the periphery of the affected areas in sections and in fresh cultures the staining is more intense and more regular than in cover-slips from the tissues. They do not stain with Gram's method. In cultures not over twenty-four hours old an imperfect staining with the method may, however,

[1] Could be tried only once on account of dying out of cultures.

be observed in such a way that some of them take the stain quite well, most of them not at all. It always seems to be the largest, best developed individuals that take the stain.

Since, as I mentioned above, a true emulsion of them in water is not formed they always appear in cover-slips in the form of clusters in which the bacteria lie very close together, so much so that sometimes it is difficult to see the separating lines between them. They vary considerably in their shape; quite a good number of them appear to be perfectly spherical, others show diplococcus forms, but in all clusters one can find individuals that are decidedly rod-shaped. Threads were not found in the cultures. Here and there one sees individuals that are about twice the normal size, oval in shape, sometimes with slightly pointed ends. These larger forms take the stain very deeply. They are more numerous in old, pure cultures, in which the smaller forms stain hardly at all any more. Spores were not found in any of the cultures. On the culture media they often die out very quickly, but sometimes they remain alive for a considerable time. In one case I succeeded in obtaining a new culture from a culture on Löffler's serum that was about two months old. Naturally, I had to take a good deal of material, and the first growth was not very abundant.

In the sections the bacteria decolorize completely when the sections are treated with either Gram's method or Weigert's fibrin stain. The best results were obtained with sections stained in dilute (1 : 5) carbol-fuchsin and decolorized in alcohol. The bacteria in the periphery of the necrosis and in the tissues next to them have very much the same appearance as those in the pure cultures. Short threads, however, are quite frequently seen. Other bacteria could not be found; there were especially no cocci to be seen in sections from rectum, liver, and lungs, which were stained with Gram's method.

The following animal experiments were made December 20, 1900: Guinea-pig. Subcutaneous injections of about ½ c.cm. of a suspension of a culture on Löffler's serum in a little beef-tea. No swelling or redness observed at point of injection at any time. The guinea-pig was killed February 1, 1901. No lesions were found.

On December 20, 1900, another pig was inoculated intraperitoneally with same amount of same material. No symptoms of disease. Autopsy, February 1, 1901, negative.

January 18, 1901, rabbit. Intravenous injection of about 1 c.cm. of suspension from Löffler's serum culture. No local changes at point of injection. Autopsy, February 1, 1901, negative.

Further experiments could not be made on account of the dying out of the cultures.

Our case, then, is one of primary infection of the rectum with sloughing and ulceration, and secondary infection of the liver by way of the portal system. It is of some interest to note, in the first place, the absence of macroscopical septic thromboses in the hemorrhoidal plexus, and, secondly, the fact that although the infection was very low down in the rectum the infectious material was carried into the portal circulation.

It seems probable that the infection of the right lung is secondary to the septic thrombosis in the hepatic veins, an embolus having been

carried away from the latter and lodged in one of the branches of the pulmonary artery.

The infectious agent I believe to be the bacterium which was found in such large numbers in the diseased tissues, and which was obtained in pure culture from the rectum, liver, and gall-bladder.

Animal experiments, it is true, have been so far perfectly negative, but they have not been on an extensive enough scale to prove the absence of pathogenic power in these bacteria for animals, and even if they should be perfectly innocuous to animals we should not be justified to draw the conclusion that this was also the case in regard to man. On the other hand, the relation of the bacteria in the tissues to the lesions is so eminently suggestive that to my mind it almost amounts to a positive proof of their etiological importance in this case. I am not inclined to attribute much importance to the few streptococci that were found in the liver and kidney, because they could not be demonstrated in the sections.

As far as the bacteria themselves are concerned, I have as yet not been able to identify them with any that have been described before. In consideration of their morphology—they being very minute, non-motile bacilli that do not form any spores, and also in consideration of the fact that they do not stain with Gram's method—they would appear to belong to the group of the influenza bacillus. From the influenza bacilli themselves they differ in growing on media that do not contain any hæmoglobin. They also differ in essential details from other members of this group that we find described in Kruse's article on bacilli in Flügge's micro-organisms (third edition, Leipzig, 1896, p. 435, viz.: B. pseudoinfluenza, R. Pfeiffer, *Zeitsch. f. Hygiene und Infectionskr.*, xiii.; B. cavernæ minutissimus, R. Pfeiffer und Beck, *Deutsche med. Woch.*, 1892; B. salivæ minutissimus, B. conjunctivitidis, Gaffky, *Arb. aus d. kais. Gesundheitsamt.*, iii.; B. pseudoconjunctivitidis, B. aeris minutissimus).

There seems to be a very close resemblance between our bacteria and the bacteria that have been described in the sputum in cases of whooping-cough by a number of authors in this country and in Europe. The identity or non-identity of the bacteria found by different authors in this disease has been a matter of much controversy. In reading the reports one certainly gets the impression that all of them have seen the same bacterium in the cover-slips prepared from the expectoration of the patients, whereas, there may be some doubt as to whether all succeeded in obtaining pure cultures of them.

Minor differences, which have played a very important rôle in this controversy, should, however, not be over-emphasized in view of the natural tendency to variation which we observe in all bacteria, especially when they are cultivated on artificial media.

The bacteria described in this paper morphologically correspond in all respects with the photomicrographs of Zusch and Vincenze, whereas they differ a little in their appearance from the photomicrographs of Czaplewski, which, however, as he himself points out, may be due to his having photographed specimens from cultures that had grown on artificial media for long time.

As far as the cultures are concerned, the only essential difference between our bacterium and the ones described in whooping-cough (as far as one is able to judge from the conflicting reports of different authors) seems to be their failure to grow on gelatin, which, after all, may be due to the fact that the gelatin was kept at a rather lower temperature (about 15° C.) than is customary in most laboratories.

Provisionally, I should recommend to call the bacterium described in this paper B. influenzæ similis.

The interpretation of the clinical history is somewhat difficult. It seems certain that when first seen the patient was suffering and had been suffering from an attack of pneumonia, which cleared up within the usual time.

The icterus which accompanied the pneumonia was at the time looked upon as a catarrhal icterus, which was probably correct. At the autopsy there was evidence of an inflammation of the mucous membrane of the duodenum, but no other causes for an obstruction of the bile ducts could be discovered.

The abscesses of the liver show all the characteristics of a very recent infection. It seems very unlikely that they should be three weeks old, and, therefore, we cannot ascribe the jaundice that existed that long, to them, quite apart from the fact that none of them interfered with any of the large bile ducts.

The rectal ulceration seems to have developed insidiously without making any symptoms quite separately from the main disease, and it seems probable that the sudden rise of temperature on December 9th, five days before the patient died, marks the time when the secondary infection of the liver occurred.

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SARCOMA OF THE LARGE INTESTINE.

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Most of the literature on sarcoma of the bowel and all of the statistical papers on the subject have appeared within the last twenty years. It is probable that, as in the case of sarcoma of other regions, notably of the mediastinum, many of the cases of this rare and interesting disease were classified as carcinomata before the era of accurate microscopical diagnosis. A number of papers on the subject have been published within the last two decades, in which the authors have gradually collected from the literature the scattered cases of this affection.

In 1883 A. Debrunner's[1] inaugural thesis was published at Zurich. In 1884 E. Pick[2] published his case. G. Newton Pitt,[3] in 1889, in a report of a case of lymphoid adenoma of the stomach and intestines, published abstracts of eighteen other cases. (These cases would be included by many pathologists under lymphosarcoma, and they are, therefore, referred to in this connection.) In 1890 Ludwig Orth,[4] in an inaugural dissertation, collected eleven cases of intestinal sarcoma, three of which were of the small and four of the large intestine, and four of the rectum.

M. Baltzer's[5] paper, which is so frequently referred to, and which was the most important and thorough study of the subject which had appeared up to that time (or, indeed, up to the present time, if we except the paper of Libman, recently published), appeared in 1892. He analyzed eleven cases from the literature and three cases occurring under his own observation in Madelung's clinic in Rostock. Smoler's[6] paper was published in 1898. Nothnagel[7] has a fairly comprehensive paper on the subject in his *System*, in the volume on diseases of the intestine and peritoneum, and Libman,[8] in his excellent article, which appeared in October, 1900, brings up to date the subject of sarcoma of the small intestine, collecting all the literature which he could find on that subject. In 1900 Blauel's[9] paper also appeared, entitled "Ueber Sarcome der Ileocecalgegend"—a collection of eight cases, including two of his own.

The operative side of the question of sarcoma of the bowel has been

recently dealt with by Van Zwalenberg,[10] who has tabulated fifteen cases of resection of the bowel for sarcoma.

Of the above-mentioned papers Blauel's is the only one which pays especial attention to any portion of the large bowel. To avoid misunderstanding, however, it must be remembered that the subject of sarcoma of the rectum is not included in the above group. The rectum is the region of the bowel which is most frequently the seat of sarcoma. Clinically it can be well separated, as a rule, from what might more properly be regarded as internal sarcoma, and, according to Paneth[11] and Rosenheim,[12] it differs very little in its manifestations from carcinoma of the same region. We have, therefore, excluded it from our consideration.

The opportunity of studying a case of sarcoma of the ascending colon from a clinical and pathological stand-point having offered itself, we have deemed it of sufficient interest, in connection with a report of the case, to collect and analyze as many other cases of primary sarcoma of the cæcum and colon as possible. To this end we have availed ourselves of a number of cases reported in the list of contributions mentioned above, and collected sundry others from the literature at large.

The following is the history of our case:

John C., aged four years, was first seen at the surgical clinic of the Children's Hospital, March 19, 1900. He was referred from the medical clinic, where he had been under treatment for some time. His history was as follows:

Parents both living and in good health. Mother had been very sickly before this child was born. One other child, an older sister, living and well. The patient, a handsome, fair-haired boy, had no sickness of note until about a year ago. He had had an inguinal hernia for two and a half years. One year before, in 1899, while in England, he had been very ill, the exact nature of the malady being obscure, except that he presented a prominence of the abdomen (which is still present), and the physician told his mother that the liver was enlarged. For several months past he has suffered from dyspeptic symptoms, has been losing flesh, and is constipated at times. An examination of the patient showed him to be a rather anæmic child, of normal stature, and not much emaciated. The physical examination gave negative results, until the abdomen was reached. This was prominent, and palpation at once revealed the presence of a rather hard mass occupying the anterior median aspect, apparently filling up the umbilical and epigastric region, and, as far as could be determined, not continuous with either liver, spleen, or kidneys, there being a zone of resonance around it which separated it from the areas normally occupied by these organs. The mass gave the impression of being under or near the anterior abdominal wall, with no intestine separating or coming between them. There were no evidences of ascites.

The diagnosis lay apparently between a tuberculous peritonitis and some form of abdominal tumor. Dr. Samuel Ashhurst examined the case with us, and it was determined to put the child on medical treat-

ment, with the possibility of the condition being a tuberculous one, and to keep him under observation for a time before proceeding to an operation. Inunctions of mercurial and belladonna ointments, equal parts, were ordered, with iron and cod-liver oil internally. The patient was not admitted to the hospital.

A few days later, March 26th, the patient developed a well-marked case of measles, which precluded all thoughts of operation for the time. The urine was examined and found to be negative, the only note being of the presence of abundant amorphous urates. Following the attack of measles, the distention of the abdomen became much greater. On April 1st it was noted that, owing to rigidity of the abdominal walls, the examination of the abdomen was unsatisfactory. There was resonance over the entire surface, and the xiphoid cartilage was pushed forward, protruding beneath the skin.

Following this date a febrile temperature developed, varying from 100.5° to 102.5°, and being highest in the evening. The tympanitic distention of the abdomen became so pronounced as to embarrass respiration, the abdominal walls being rigid, the veins dilated, the umbilicus pouting, but with no ascites. No obstruction of the bowels was present. Food seemed to cause discomfort, and there was evidently abdominal pain or at least discomfort at other times. Enemata of turpentine and the passage of a catheter were resorted to in order to relieve distention, and on April 8th the child passed, after an enema, a piece of fleshy tissue, about four inches long and less than half as wide, which resembled bowel wall, being thick, of fleshy consistence, and dark red in color. Under the microscope it seemed to be composed of fibrin, but, unfortunately, no careful histological examination of it was made.

Following this the temperature remained elevated for about two and one-half weeks, while emaciation became more and more marked, and the difficulty in feeding increased. The distention, which had been so extreme, became gradually reduced, being apparently lessened by the use of inunctions of mercurial and belladonna ointments and the application of a tight binder. The child was very peevish and resisted examination.

By May 5th the temperature had remained normal for some days, but weakness and emaciation were progressive. He had vomited a great deal for two or three days, and complained of pain at the nape of the neck. There was a somewhat troublesome cough, but no definite pulmonic signs. The bowels moved naturally. There was still some distention of the abdomen at the costal margin, but not much below this. The mass could be again distinctly outlined, but now in the right iliac region. The detection of the tumor had been impossible while the distention was so pronounced.

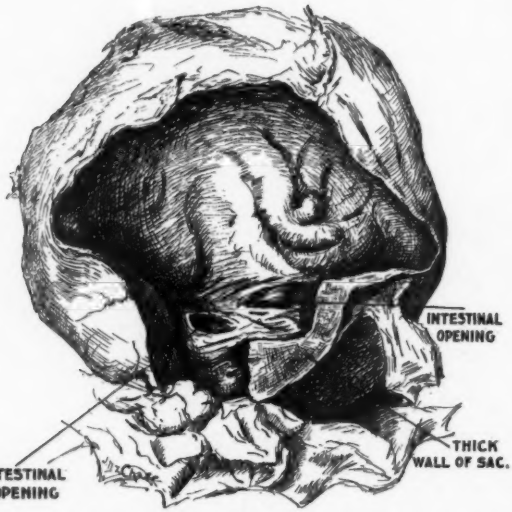
The child now refused almost all food and medicine. A few days later the left foot and leg became swollen and œdematous, and he developed what the mother described as convulsions—spells accompanied with crossing or rolling upward of the eyes, movements of the arms and legs, and followed by coma. Several of these occurred in the twenty-four hours. Examination of the urine showed only a small amount of albumin. Occasional movements of the bowels were noted. On May 14th, after having vomited everything for some days, and with no new symptoms, he died, apparently of asthenia, being emaciated to the last degree. At no time after the unfortunate attack of measles

was he in condition to undergo laparotomy, which both the parents and ourselves looked forward to as offering a definite solution of the diagnosis, and perhaps some hope of relief.

Pathological Notes. The autopsy was performed twenty-four hours after death, and the following notes made:

The body is emaciated in the extreme. Rigor mortis is only partially developed. The left leg, foot, and thigh are moderately œdematous; no scars; slight discoloration in the dependent portions of the body. Incision in the median abdominal line reveals a very slight amount of subcutaneous fat. The incision opens up a cavity in the shape of a sac extending from above the umbilicus nearly to the symphysis pubis. Laterally it extends only slightly to the left of the median line, while

FIG. 1.



Sarcoma of the large intestine, showing aneurismal sac with the intestinal openings on the posterior wall.

on the right it reaches nearly to the anterior superior spine of the ilium. The walls of this sac are adherent to the anterior wall of the abdomen, and at this place are about 2 mm. in thickness. To the right and posteriorly they increase in thickness; the greatest thickness is about 2 cm., being at the posterior portion of the sac. The walls of the sac are composed of a yellowish-white, homogeneous tissue, of rather firm consistency, the external surface being mainly smooth, except in places here and there, especially posteriorly, where it is somewhat nodular. The inner surface is rather granular. The capacity of the sac is about one pint, and at the autopsy it contained about twelve ounces of a turbid, yellowish fluid, devoid of any special odor.

On examining the inner surface of the sac the posterior wall is seen to present two openings, which correspond with the entrance and exit of

the ascending colon. In the space between these two openings, which is about 5 cm. in length, no naked-eye vestiges of the former bowel can be found, the mucous membrane stopping rather abruptly on the surface of the thickened area at the openings of the bowel. Externally the sac is situated about 18 cm. above the ileocæcal valve, is pear-shaped, the base completely surrounding the ascending colon, and the apex pointing toward the median line. The only attachment of this sac to the parietal peritoneum is in the median line of the abdominal wall along the line of incision. After removal the sac measured 14 cm. in its vertical by 11 cm. in its transverse diameter.

The remainder of the peritoneal cavity in the immediate vicinity of the sac is free from adhesions. From the tumor, along the mesenteric attachments of the bowel, both above and below the sac, there is much infiltration, the mesentery being in places a full centimetre in thickness. The rest of the abdominal cavity seems quite free from involvement, excepting for a false diaphragm which stretches below the liver, stomach, and spleen, forming a separate cavity for these organs. There is an apparent perisplenitis and perihepatitis which attaches these organs to the diaphragm.

The spleen is not enlarged; the capsule is thickened and in places covered with a distinct yellowish-white exudate. The organ is firmly attached to the diaphragm by dense adhesions. On section the surface is brownish-red in color, and slightly fibroid in character.

The liver is greatly enlarged, of soft consistency, and of a bright yellow color. The outer surface is smooth, with the exception of several small areas which present a yellowish-white exudate, and in a few places it is adherent to the diaphragm. On section it is almost bloodless, and of the same bright yellow color throughout. The gall-bladder is normal.

The organs of the thoracic cavity are to all appearances normal, with the exception of one mediastinal lymph gland, which is distinctly enlarged, and on section is homogeneous, of a yellowish-white color, and of rather firm consistency. There is also a slightly enlarged lymph gland on the thoracic side of the right half of the diaphragm. The lungs show hypostatic congestion, but no evidence of tubercle or new growth.

The kidneys are very much enlarged, the left, which is the larger of the two, measuring 10 cm. in length by 5 cm. in width, and 4½ cm. in thickness. The capsule strips easily. They both present a slightly lobulated appearance, with irregular nodular prominences, of a yellowish-white color. The superficial vessels of the cortex stand out quite prominently on this yellowish-white background. On section the surface presents yellowish-white areas, especially prominent in the cortex; these areas are diffuse in places, and in other parts distinctly circumscribed. There are no areas of softening, and the bloodvessels, especially of the medulla, are prominent. There seems to be very little of the normal kidney substance remaining.

The suprarenal glands are not changed in appearance. The stomach appears normal. The small intestine is also normal. The cæcum and the appendix are infiltrated, especially along the mesenteric attachment, with a direct continuation of the growth. The retroperitoneal and mesenteric lymph glands are enlarged; some show the yellowish-white appearance so characteristic of the tumor mass, while others are red and soft.

The sac, with the cæcum and ascending colon, and a part of the ileum, were removed intact, as were also pieces of the liver, spleen, and left kidney, and much of the mesentery, and the enlarged retroperitoneal and mediastinal lymph glands. They were fixed with Müller solution, formol, etc., for further study.

Microscopical Examination. Sections were made from the thickest portion of the sac at its base; from its thin upper portion; from the posterior wall, including the afferent and efferent openings of the colon; from the appendix, and the lymphatic glands, peritoneal and mediastinal; from the spleen, the kidney, and the liver. Sections from the thickest portions of the sac are covered on the outer surface with peritoneum, and are composed of a reticular fibrous network, aggregated here and there into denser masses of connective tissue. The meshes of this framework are densely packed with small, round, mononuclear cells, the nuclei of which take the stain moderately well and show very distinctly a chromatin network, some also showing evidences of karyorrhexis, and here and there mitotic figures. Surrounding the nucleus is a very small rim of protoplasm. In the denser portions of the connective-tissue framework are bloodvessels of moderate size, with fairly well-formed walls. The rest of the tumor contains small capillaries with distinct walls. Near the inner surface of the sections of the thick portion of the sac are vestiges of smooth muscle fibre, which are only recognizable as bundles in a few places, owing to separation of the individual fibres by the infiltration of the round cells previously described. This area of muscular tissue remaining is quite close to the internal surface of the sac, showing that the mucous membrane and submucosa have sloughed away.

The thin portion of the sac in relation with and adherent to the anterior abdominal wall is covered with peritoneum, and shows itself on section to be composed of two layers—the outer is composed of fibrous tissue, with few nuclei among the connective-tissue bundles; the inner, made up of fibrous tissue infiltrated with chains of the peculiar round cells described. The walls of the bloodvessels here are also well formed. The juncture of these two layers is more or less defined, though in places the infiltration to the fibrous layer is quite evident.

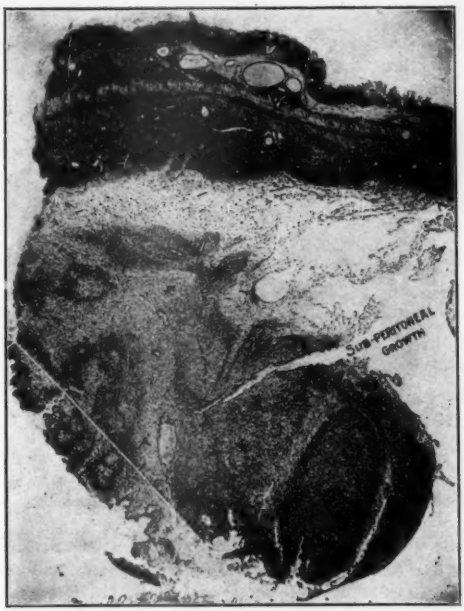
A longitudinal section taken from the posterior wall of the sac, between and including a portion of the afferent and efferent parts of the colon, in connection with the sac, is covered with peritoneum. At each end of this section the bowel is about normal in appearance, excepting for a very slight subperitoneal infiltration of the tumor cells. As we approach the centre of this section, a point corresponding with that portion of the sac between the ends of the bowel, the layers of the intestine become more and more separated, and their appearance altered by the increasing infiltration of the new cells. On the mucous membrane the tubules become shorter and further apart, and, finally, rather abruptly disappear. The muscularis mucosa gradually disappears, but the bloodvessels seem to become more numerous and greatly dilated. In places they have an angiomatous arrangement. The muscular layer, as above mentioned, comes near the surface as the other layers disappear. The individual bundles are greatly infiltrated, so that in the middle of the thickened area only an individual fibre is occasionally seen.

In the region where the sac springs from the colon, and where the tumor infiltration completely surrounds the bowel, the new growth

pushes its way between the two layers of the mesentery. Along the cæcum this makes a partial mantle, and on reaching the appendix completely surrounds the entire length of this organ. This surrounding mantle is in places thicker than the intestinal wall itself. The appendix is infiltrated in all its coats, to such an extent in some sections that the normal structure is scarcely to be recognized. In one of these sections an area of degeneration resembling caseous material is to be seen. This is the only area of degeneration found in the tumor.

The lymphatic glands of the mesentery in the region of the tumor are enlarged, of moderate size and consistency, and on section they

FIG. 2.



Section of bowel wall, with subperitoneal growth.

show a homogeneous structure of whitish color. Microscopically the capsule of the gland and the fibrous trabeculæ show distinct thickening; in places the capsule is distinctly infiltrated with the small round cells, and in some glands there is a mantle of round cells surrounding the gland.

The fatty tissue of the mesentery between the glands shows tracts and heaps of small round cells.

The same structure was observed in the case of the isolated enlarged gland of the mediastinum.

Spleen. The capsule and trabeculæ are distinctly thickened; the Malpighian bodies appear to be slightly enlarged, but they are not

distinctly outlined. What was at autopsy thought to be a perisplenititis is found to be a deposit of small round cells similar to those of the tumor.

Kidney. The nodules observed arising from the surface of the kidney are composed of almost pure tumor tissue. They exceptionally show an altered glomerulus, the only remains of the former kidney substance. Between these nodules the kidney structure is more in evidence, although the tubules are separated by the infiltrating tumor cells. The cortex is greatly thickened, this thickening being due to the new growth. The medulla is less involved, and the tubules of the pyramids in many places show only slight changes. The tubules, especially in the cortex, show cloudy swelling, fatty degeneration, necrosis, and in places desquamation of the lining epithelium.

FIG. 3.

Section of kidney, showing glomerulus and infiltrating tumor cells.

Liver. The sections of the liver present a remarkable degree of fatty infiltration and degeneration; in fact, these changes are so pronounced that a normal liver cell can scarcely be seen. Along the interlobular connective tissue, especially surrounding the vessels and ducts, there is a beginning infiltration of the new growth.

Nearly all of the writers on sarcoma of the intestine have prefaced their remarks with comments upon its rarity. Statistical proofs of this are easily found. According to Baltzer,[5] Stort found no cases of sarcoma of the bowel in the records of the Berlin Pathological Institute from 1859 to 1875. Baltzer analyzed fourteen cases, three of his own and the rest collected from the literature. Nothnagel[7] dwells on the comparative infrequency of intestinal sarcoma compared with carcinoma. In the Wiener Allgemeinen Krankenhause, from 1882 to 1893, of 2125 autopsies on cancer cases 243 were of the bowel. Between the same periods, of 243 sarcomata only 3 were of the bowel. He

quotes Müller, who found in Berne, of 521 cancers, 41 of the intestine, and of 102 sarcomata but 1 of the intestine, the ileum. Smoler,[6] in fifteen years, from 1883 to 1898, out of 13,036 autopsies found 13 cases of primary sarcoma of the small intestine—a proportion of 1 to 1000. Libman was able to collect about 59 cases in all of sarcoma of the small intestine.

When we compare the relative frequency of sarcoma of the large with that of the small intestine, we find it universally admitted that the latter constitutes the larger class numerically. Disregarding for the present the difference between lymphosarcoma and sarcoma, which, as Nothnagel says, while pathologically distinct, cannot be separated clinically from each other in this locality, we are able to collect the following data: Of 12 cases mentioned by Nothnagel, 1 was duodenal, 3 in the jejunum, 4 in the ileum, 3 in the cæcum, and 1 in the rectum. Nothnagel remarks on the difference between sarcoma and carcinoma of the bowel, the favorite seat of the latter being in the large intestine. This difference is present, he says, at least in the case of lymphosarcoma, to which class 9 of the above tumors belonged. Of Smoler's 13 cases, 2 are included in our list as probably primary in the large intestine, although he includes them in his table of small intestine cases. Treves[13] says that lymphosarcoma is rare in the colon alone, and much more common in the small than in the large intestine. Of 18 cases, in 5 the tumor was located in the stomach, small intestine, and colon; in 1, in the stomach and small intestine; in 3, in the small intestine and colon; in 8, in the small intestine alone, and in only 1 case in the colon alone. He gives the ileocæcal region as a common seat, and states that the tumors grow largest in this locality. Excluding the rectum, he gives the small intestine as the most common site of sarcoma, which, he states, is very rare in the colon. The relative frequency is given by Rosenheim[12] as first in the rectum, and next in the small intestine. Of Blauel's[9] 8 cases of sarcoma of the ileocæcal region we have included 6 in our table as primary in the cæcum. Of 10 cases of sarcoma of the intestine studied by Ablon,[14] 2 were in the large bowel. Libman states that sarcoma by preference occurs in the small intestine, and that in the large intestine (always excepting the rectum) they are much less common, and he quotes Krueger as follows: Of 38 cases, 16 were in the small intestine, 1 in the ileum and cæcum, 2 in the cæcum, 1 in the appendix, 1 in the transverse colon, 1 in the small and large intestine, and 16 in the rectum.

Libman collected 59 cases of sarcoma of the small intestine; we have found in our search but 22 cases of sarcoma of the large intestine. We have analyzed these cases as far as the data we could secure permitted, with the following results:

Age. The age of the patients ranged from under two years to over sixty. There were seven under ten years; certainly two and probably

three between ten and twenty years; four between twenty and thirty; five between thirty and forty; one between forty and fifty; one between fifty and sixty; and one over sixty. The first decade contained the greatest number; the fourth decade the next greatest. There were only three cases past forty years of age.

SEX. The cases were nearly equally divided in point of sex, there being 12 males and 10 females.

LOCATION. Of the 22 cases which we have tabulated as primary sarcomata in the large bowel, in 5 the primary location was open to some doubt, there being, however, a strong probability that it was in the large intestine. In 2 of these there was involvement of the last portion of the ileum. In 1 case—that of Lange—no other part of the intestine was involved, but the growth may have been primary in the ovaries, which were sarcomatous. In 3 other cases, while primary in the large bowel, it involved the ileum by extension. In the remaining 14 cases the large bowel alone was the seat of the sarcoma. When we study the location of these tumors more in detail we find that in 7 cases they were confined to the cæcum (including with it the ileocæcal valve and the appendix); in 5, the cæcum and the ileum were affected; in 3, the cæcum and ascending colon; in 4, the transverse colon; in 1, the descending colon, and in 2, the sigmoid flexure. The cæcum is thus seen to be the most common seat, the next in frequency being the cæcum and ileum; the ileocæcal region comprises the seat of 12 out of 22 cases, or 54.5 per cent. The transverse colon stands next, being the primary seat in 4, or 18.1 per cent.

SIZE. In 8 the size was not stated; in 1 it was said to be the size of a man's head, while in 3 it was the size of a child's head; in 1 it was compared to a cricket-ball. Briefly, the tumors ranged in size from that of a hen's egg to that of a man's head. In one case the growth was in the form of a ring (causing partial obstruction).

VARIETY. In tabulating the tumors from the morphology of the cell we have preserved separate columns for small round-cell sarcoma and lymphosarcoma, although it is difficult to make any definite boundary between these two classes. Where the reporters have described them as such, or from their histological examination they have seemed to us to fall in this group, we have included them as lymphosarcoma; where they have been described as small round-cell sarcoma we have classified them under this head. In 2 cases the type of cell was not stated. Of the remaining 20, 10, or 50 per cent., were round-cell sarcoma; 9, or 45 per cent., were lymphosarcomata, while 1 was of the spindle-cell variety.

METASTASIS. Of 19 cases in which the data as to metastasis was given the abdominal lymphatic glands were involved in 13, or 68.4 per cent.; the peritoneum in 5, or 26.3 per cent.; the small intestine (by metastasis, not by extension) in 2, or 10.5 per cent.; the lung in 2, or

10.5 per cent.; the kidney in 2, or 10.5 per cent.; the spleen in 2, or 10.5 per cent.; the liver in 3, or 15.7 per cent.; other glands in 2, or 10.5 per cent.; the epididymis and spermatic cord in 1; the mesentery (subperitoneally) in 1; the orbit in 1, and in 1 case there was a secondary growth in the large intestine. In 1 case there was no metastasis, and of the 3 cases in which the data was wanting 1 *case recovered after operation.*

The abdominal lymphatics are thus seen to be by far the most common sites for the deposit of metastatic growths, with the peritoneum next in point of frequency. Except where the peritoneum is involved by continuity the road of dissemination of the infecting cells is through the mesentery, and this would explain the frequent and early involvement of these glands which was present in many of the cases at autopsy and in those subjected to radical operation.

Etiology. The etiology is as obscure as is the case in internal malignant diseases elsewhere. In at least two of our cases there was a strong family predisposition to malignant disease, and there was a history of abdominal traumatism of doubtful influence in two others. Flexner[15] and Libman[8] have called attention to peculiar bodies which they detected in sections from lymphosarcomata of the stomach and small intestine, which they regarded as possibly standing in the relationship of causative organisms.

Origin of Tumor. The place of origin of the above tumors is rather difficult to locate on account of the size of the tumor and the involvement of the bowel when the subject presents itself either to the operator or to the pathologist; but from the data of the microscopical examination of the tumors which we have been able to collect, and from the careful examination of our own case, it seems to us that the mucosa or submucosa is the starting-point of these tumors, and, from the normal histology of the intestine, either may be the origin if we take for granted that they have their origin in the lymph follicles. From the mucosa or the submucosa they involve the other coats—the muscular offering the greatest resistance, and, though always infiltrated, traces of this layer are usually found, sometimes as the only remains of the former gut. The subserous coat, from its position, offering very little resistance, we find the greater mass of the tumor usually between the peritoneal covering and the remains of the muscular coat. The serosa itself is rarely perforated. This corresponds with what Baltzer pointed out years ago as the behavior of these tumors in the small intestine. The absence of stenosis and the frequent occurrence of dilatation, sometimes even aneurismal in character, as in our own case, has been explained in the small intestine by the early infiltration and paralysis of the muscular fibres, and the consequent dilatation by accumulation of feces. However, the early involvement of the submucosa, with its plexus of nerves, must not be forgotten as a potent factor in this dilatation.

The *alterations* produced in the segment of bowel involved may be classified as, first, involvement of the whole circumference of the bowel in a rather limited space, forming a rounded tumor with or without dilatation of the lumen. This is the most common condition. The dilatation may be actually aneurismal in extent, due to stretching of the infiltrated wall or to extensive ulceration and breaking down of the interior of the tumor. In about 20 per cent. of these cases the tumor was situated only on one side of the gut. Again, the bowel may be converted into a thick-walled tube, the infiltration extending over a considerable distance in the bowel. In the transverse colon the tumor tends to proliferate in the great omentum, the bowel losing itself in the mass. In only one case was the tumor of the narrow, ring-shaped, stenosing type often seen in carcinoma.

DEGENERATION. The common degeneration of the tumor consists of ulceration of the mucous membrane over the tumor mass, softening within the tumor mass, and subsequently cavity formation. This softening may lead to perforation, with direct communication to the peritoneum, and suppurative peritonitis (in one case purulent peritonitis was found without discoverable perforation), or perforation may follow adhesion between the tumor and loops of the small intestine, as in Blauel's second case (No. 18).

SYMPTOMATOLOGY. In 5 of the 22 cases little or no clinical history was obtained from the sources at our command. Emaciation was mentioned as being present in 11 cases; in 10 it was not mentioned; in 1 case it was not present. It is thus seen to be a frequent symptom, as might be expected from the nature of disease. In our case, as in several others, it was extreme. In the case in which it was not present death was due to septic peritonitis, and not to cachexia and exhaustion, which are common causes of death.

FEVER was present in 9 cases, usually a moderate elevation, especially noted before death. In 1 case, in which there was a secondary infection (probably with the bacillus aërogenes capsulatus and another organism), there was high fever, running up to 104.4° F. for the last four days of life.

ŒDEMA of both lower extremities was present in 1 case, and of the left lower extremity in our own. Pressure upon the iliac veins might, with general weakness and position, be considered as the cause. Ascites was very rarely present.

TUMOR was detected in 16 cases. It was not detected in 2, not noted in the history in 3 (although 2 of these were operated upon), and in 1 no data were obtainable (Hoffmokl, Case No. 21). The presence of a tumor is thus seen to be a most noticeable symptom.

The *locality* in which the tumor was detected during life varied considerably. In 7 it was outlined in the right iliac fossa, in 1 of these

cases there being two other tumors in other localities. In 2 it was primarily in the median umbilical region, one of these later shifting to the right iliac fossa, and the other extending over the greater portion of the abdomen. One was in the epigastric region, and four were situated to the left of the median line, one being in the left umbilical region, one in the left hypogastric region, and two in the left hypochondriac and left lumbar regions. The frequent presence of the tumor in the right iliac region corresponds with the frequent ileocæcal location of the growth. In all of the 7 cases in which the tumor was observed to be situated here, the ileocæcal region was found to be the site of the tumor, with one exception, that in which two other tumors were detected during life, in which the transverse colon was the primary site, and in which there were numerous metastatic growths. The next most common location was to the left of the median line. Of the 2 cases observed in the left umbilical and hypogastric regions, 1 was of the transverse colon, and 1 of the sigmoid flexure. Of the 2 in the left hypochondriac and lumbar regions, 1 was a cæcal tumor with intussusception of the ileum and cæcum into the colon, and one was of the sigmoid flexure. The epigastric tumor sprang from the ileocæcal region of the bowel, and here, also, there was intussusception. Of the two in the umbilical region, one—our own case—was of the cæcum and ascending colon, which afterward shifted to the right iliac region, and one was a tumor of the transverse colon. The sarcomata of the cæcal region of the large intestine are thus seen to be usually observed in the corresponding area of the abdominal surface, but may be detected in the median line, or, where there is an intussusception, in the epigastrium or left hypochondrium and left lumbar region. Tumors of the transverse colon usually occupy a median position, sagging downward; tumors of the sigmoid may be looked for to the left of the median line.

In the majority of cases in which the clinical symptoms were given the tumor was described as hard, sometimes of a smooth and sometimes of a nodular feel, rounded or flat, and in two cases there was a sense of fluctuation elicited on palpating it.

Mobility. It was more than twice as often stated to be movable as immovable, and in two cases it is noted that it moved with respiration. The liver was sometimes displaced.

The tumor was frequently found to be tender on palpation.

Gastro-intestinal Symptoms. These were mentioned in 14 cases or 63.6 per cent. They were mainly pain, anorexia, and vomiting. Pain was present in the majority of cases; in fact, in only one was it stated to be absent. Several other incomplete cases made no reference to it. In 3 it was increased on taking nourishment, and in 1 during urination. It was twice the first symptom noticed, and it was often

severe, periodical in some, acute in some, and colicky in others. It was always referred to the abdomen, but not always to the tumor.

In 3 cases it simulated appendicular pain in character and location, and in these cases the tumor was in the ileocæcal region.

Other occasional symptoms were: bloody stools, 3 cases; diarrhœa, 2 cases, and general dyspeptic symptoms were present in 9.

Obstruction. The absence of obstruction in the large majority of cases is one of the striking characteristics of this form of malignant disease of the large bowel, and is a characteristic which it shares with sarcoma of the small bowel, and which separates it sharply from carcinoma of the same locality. It was certainly absent in 13, or 59 per cent., and it was not noted in the history, and, therefore, probably absent in 6 cases. It was complete in 1 and incomplete in 1, and in 1 the clinical history was not obtainable. In 1 of the 13 cases obstruction was present once during the course of the disease, from twisting of the tumor, as the symptoms disappeared after manipulation; hence obstruction in its truest sense did not exist in this case.

In the case of complete obstruction (Horn) the seat of the tumor was in the descending colon. It was the only case of the spindle-cell type, and the obstruction was due to almost complete stenosis, with impaction of a small fecal mass. In the case of incomplete obstruction, one of Debrunner's (Case No. 4), the tumor was a ring-shaped, round-cell sarcoma of the sigmoid flexure. Thus the only tumors producing obstruction were located in the descending portion of the large bowel.

Obstruction in this class of tumors involving the small intestine has always been due to intussusception, volvulus, or adhesions. In two of our series of cases *intussusception* was found (both tumors of the ileocæcal region), and in neither did it cause obstruction.

Distention. While not a constant symptom this was observed quite frequently, being especially noted in eight cases, often in a marked degree; in one case only was it due to obstruction.

Appendicitis Simulated. In Libman's paper on sarcoma of the small intestine he reports five cases, in three of which the clinical picture was very similar to that considered diagnostic of appendicitis. He lays much stress on this important clinical observation, and remarks that in attempting to ascertain whether such resemblance had been noticed before he found that the clinical picture was an unknown one. He is not the first, however, to call attention to the fact that sarcoma of the bowel may closely simulate appendicitis. Orth,[4] in 1890, in speaking of the diagnosis of sarcoma of the intestine, dwelt upon the difficulty in separating some cases of typhlitis and perityphlitis from it. Wharton,[15] in 1892, published a paper entitled "Sarcoma of the Cæcum Simulating Appendicitis," and in the case he reported exploration was necessary to differentiate between these conditions (Case No.

11). He personally narrated to us the history of another case, in which the resemblance was as striking (Case No. 12). In one of Blauel's cases, also, is an attack of supposed appendicitis noted as preceding the discovery of the tumor. The fact that the appendix is very often the seat of infiltration in ileocæcal growths may be partly responsible for the symptoms.

There are practically no data in the cases which we have collected in reference to the condition of the blood, although the patients were often said to be anæmic. One case (Fisher, Case No. 19) gives the blood examination three days before death: Hæmoglobin, 50 per cent.; no poikilocytosis or leucocytosis. There is a similar lack of data in the urinary examinations. In our own case albumin was noted late in the course of the disease. In Baracz's case blood cells and blood casts were present in small quantity, without involvement of the kidney. In two cases the urine was normal.

Secondary Infection. In Fisher's case, where the sarcoma was located in the cæcum, the patient, a male, aged twenty-eight years, first detected the tumor three weeks before admission to the hospital. One week later he developed abdominal pain, followed by swelling of the gums and bleeding, and later by the appearance of a purpuric rash liberally scattered over the front of the body and the neck and arms. Epistaxis was present. The blood showed 50 per cent. hæmoglobin, and no leucocytes or poikilocytes. There was irregular fever during the three days he was under observation in the hospital. He died, and at the autopsy the subcutaneous tissues, especially of the neck and scrotum, and the peritoneal cavity, were distended with gas; and there were small gaseous cysts scattered thickly through the tumor and the spleen, and present in the liver, and to a less extent in the kidney. Aërobic cultures were sterile; anaërobic not made. Microscopically many bacilli were found in the growth among the sarcoma cells around the cysts, often in pairs, and showing a clear space—probably an unstained capsule—around each bacillus. He regarded this germ as probably the bacillus aërogenes capsulatus, and the infection was probably a mixed one. He refers to the case of Hilton Fagge as showing the association of sarcoma with purpura. He thinks the sarcoma acts by lowering resistance and, at the same time, providing a portal of entrance for the germ. Hilton Fagge's[17] case was one of sarcoma of the end of the ileum and the ileocæcal valve, and the symptoms bore a striking resemblance in some points to those of Fisher's case just described. It is not included in our list of cases. The symptoms were entirely those of secondary infection. After some exposure the patient suffered for several weeks from scattered pains in some of the joints, especially the hips and shoulders, without effusion, and had fever and acid sweats. When admitted to the hospital about a month later he was supposed to

have rheumatic fever. He then developed a purpuric rash, with hæmaturia, great swelling of the eyelids and scrotum, and ecchymosis of the conjunctivæ. Nodules appeared at the sites of some of the purpuric spots. After death, about two months after the onset of the symptoms, decomposition rapidly set in, and sixteen hours later, at the time of an autopsy, was quite advanced. The scrotum, thighs, and upper arms were emphysematous. The nodules in the skin proved to be sarcomatous.

Duration. It is difficult to estimate the duration of the disease in this class of cases, as in all of internal malignant disease where the history and the symptomatology have to be depended upon, for the new growth is undoubtedly present for some time before the patient or the physician discovers it. Hence the duration can be estimated only from the time of onset of the symptoms. In the cases not subjected to radical operation, the duration of life varied from one to eighteen months; in the cases in which a radical operation was performed, the duration of symptoms varied from one week to five years before operation, although in the latter case the tumor had been observed for only eight months. One case not operated upon gave no definite symptoms until obstruction developed a few days before death.

The usual causes of death in non-operative cases were cachexia and exhaustion. These were noted in at least six cases—in one, associated with recurrence after the operation. In another case with wide-spread metastasis the cause of death was given as heart failure; this was also probably due to exhaustion. Two cases died of purulent peritonitis, probably due to infection from the bowel; in one of these cases there was incomplete obstruction. One case died of acute obstruction, and another from mixed infection (purpura hemorrhagica).

Fourteen of the 22 cases were subjected to operation. In 4 cases the scope of the operation was purely exploratory, no attempt at removal being carried out. In 10 cases resection of the bowel was performed; 5 recovered from the operation, and 5 died as a result of it—a mortality and recovery rate of 50 per cent. each. Of the 5 cases that recovered 1 died of recurrence after forty-seven days. In 1 no data, beyond the fact of recovery, were attainable; 1 was well after six months; 1 after seven months; 1 after three years. The last case (Orth's) had been operated on five years previously for sarcoma of the ovaries; while the growth in the bowel may have been secondary to that in the pelvis, extirpated five years before, it is included here because of the length of time intervening, and because no other portion of the bowel was affected. In at least 4 of the 5 successful cases enlarged lymphatics were removed from the mesentery; and the fact that these cases recovered shows that the prognosis need not be bad on this account alone, and also the advisability of searching for and removing them.

Diagnosis. The diagnosis of intestinal sarcoma, as of all intra-abdominal tumors, is a matter of much uncertainty. As Osler says, it

is here that Bishop Butler's maxim that "probability is the rule of life" finds true application. Of 14 of our cases in which we have recorded the diagnosis made before operation or autopsy, in only 2 can the tumors be said to have been fairly accurately localized; in Bouilly's case a diagnosis of malignant tumor of the intestine having been made, and in Orth's, one of mesenteric tumor. They were taken for tumors of the kidney, the omentum, the ovary, the peritoneum, the lymphatic glands, and in two cases they strongly simulated appendicitis—an observation of considerable importance. Again, the presence of metastasis to the lymph glands and the peritoneum may give rise to the presence of multiple tumors, which may be very confusing. We will briefly summarize the prominent symptoms presented in our cases, and then compare them with those offered by sarcoma of the small bowel and by carcinoma of the intestine.

The primary symptoms are gastro-intestinal manifestations of a dyspeptic type, especially abdominal pain, anorexia, and vomiting; progressive, rapid emaciation; moderate fever; abdominal distention, usually without ascites, but occasionally associated with œdema of the lower extremities; and the presence of a tumor, which is more often movable than immovable, frequently tender, usually of firm consistence, and often located in the right iliac region. The absence of obstruction in the great majority of cases is of great significance. The above chain of symptoms occur in persons of any age, but with greatest frequency in those under forty years, and with relative frequency in the early years of life. The group of symptoms cited does not differ materially from that associated with sarcoma of the small intestine, except, perhaps, the more frequent location of the tumor in the right iliac fossa in disease of the large intestine, owing to the frequent location in the cæcal region. The tumor may be very tender in the case of the large intestine, in which it probably does not differ from some cases of tumor of the small bowel, which in the majority of instances, however, are said to be slightly or not at all tender. Both are liable to be complicated by intussusception. König[18] believes invagination in intestinal tumors to be more common than is usually supposed, citing two cases in discussion.

Libman's statistics show that in the small intestine sarcoma may be found at all ages, although a large percentage of his cases were past thirty. A peculiar paleness or lividity of the complexion has also been described as rather characteristic of cases of sarcoma of the small intestine by both Baltzer and Libman. The differential diagnosis between this tumor of the intestine and carcinoma of the bowel, as well as between it and other intra-abdominal tumors, will, of course, arise. The first question is the only one we need refer to here. Baltzer laid stress on the age, sarcoma occurring earlier; the more rapid growth of sarcoma; the absence of obstruction, of marked tenderness, and of pain during defecation; the early appearance of cachexia and the much

greater prominence of general over local symptoms. Libman believes carcinoma of the bowel to be more common than Baltzer supposes in early life, but grants that under fifteen years the chances are in favor of sarcoma; he dwells on the absence of external glandular involvement, the absence of tenderness, and the large size of the growth in cases of sarcoma.

The main points of differentiation would seem to be in a sarcoma of the large bowel, its appearance in the majority of cases in early life (under forty), the early appearance, rapid growth, and large size of the tumor, the quick cachexia, and the absence of obstruction in most of the cases. Pain and tenderness may be present in both; but pain is an early and rather constant symptom in sarcoma of the large intestine, while in carcinoma it does not usually manifest itself until symptoms of obstruction develop.

Prognosis and Treatment. The prognosis without operation is, of course, bad. The life of the patient may be prolonged through a longer or shorter period of suffering, but medicinal treatment of specific nature remains to be discovered. Libman urges the use of arsenic where resection cannot be practised, calling attention to the fact that genuine cases of lymphosarcoma, usually multiple, or of the skin, have been cured by its exhibition, either internally or by injection. We know of no cases of intestinal sarcoma so relieved. The same is true of the use of Coley's fluid. On the other hand, operation, with resection of the growth, offers a fair chance for recovery in cases taken sufficiently early, the prognosis being at least as good, once the tumor can be safely removed, as in sarcoma elsewhere. It is contraindicated in cases far advanced in cachexia, which would be unable to stand the shock of operation, or where there is any other reason to believe that the widespread metastasis so characteristic of the later stages is present. In cases seen early, and in doubtful cases, exploratory laparotomy should be undertaken; 14 out of our 22 cases were operated upon. In 4 the operation was simply exploratory, no attempt being made to remove the tumor. In 10 cases resection was practised; 5 of these died as a result of the operation, either of shock or of peritonitis. Of the 5 that survived the operation, 1 died in forty-seven days of recurrence. Here, certainly, some metastatic growth had been present and not detected at operation. Of the remaining 4 all were living at the time of reporting. This was six months later in 1 case, seven months later in 1, three years in 1, and not stated in 1. In at least 4 of these 5 cases, as has already been remarked, enlarged mesenteric glands were removed at the operation, showing that the presence of metastasis to the lymphatics is no contraindication to operation, and also the necessity of searching for these glands and removing them, if a radical operation is to be performed.

TABLE OF CASES.

Reporter.	Sex and age.	Variety.	Location.	Duration.	Metastasis.	Operation.	Remarks.
1. H. Arnott, Trans. Path. Soc. London, 1874, xxv.	M. 4 yrs.	Lympho-sarcoma (lympho-ma).	Transverse colon.	A little over 2 months.	Mesenteric glands, kidney, liver, epididymis, spermatic cord, skin of abdomen.	None.	Died from purulent peritonitis.
2. W. Horn, Hamburg, 1882, quoted by Orth, loc. cit.	F. youth.	Spindle-cell sarcoma.	Descending colon; stenosis of gut.	Unknown; no symptoms before obstruction developed.	Not stated.	None.	Death from acute intestinal obstruction due to stenosis of intestine.
3. A. Debrunner, Inaug. Dissert., Zurich, 1883, quoted by Orth	M. 5 yrs.	Round-cell sarcoma.	Transverse colon.	About 7 weeks.	To omentum, whole abdominal cavity seem'd filled with white or pale red tumors.	None.	Death from exhaustion.
4. A. Debrunner, loc. cit.	F. 16 yrs.	Round-cell sarcoma.	Sigmoid flexure. Some stenosis.	4 months.	Retroperiton'l glands, left ovary, apex of left lung.	None.	Death from peritonitis.
5. H. Beck, Zeitschr f. Heilkunde, 1884, v. p. 442.	F. 59 yrs.	Lympho-sarcoma.	Cæcum and ileum.	Not stated.	Cervical lymph gl'nds, both lungs, encroaching on pleura and pericardium, spleen, stomach, ileum, and mesenteric glands.	None.	Diagnosed lymphoma of cervical and abdominal lymphatic glands; death from heart failure.
6. F. Lange. New York Med. Journ., 1886, xliii. p. 199.	F. 32 yrs.	Not stated.	Transverse colon.	Uncertain.	Omentum, mesenteric glands, both ovaries.	Resection of bowel, removal of ovarian tumors.	Primary seat doubtful; possibly in ovary. Death from septic peritonitis on ninth day; leakage probably from incessant vomiting.
7. Bouilly, Bull. et Mém. de la Soc. de Chir. de Paris, 1888, n. s., xiv. p. 601.	F. 44 yrs.	Lympho-sarcoma.	Cæcum and ileum.	Symptoms for 5 years; tumor for 8 months.	Lymphatic glands in mesentery.	Resection.	Recovered; well seven months later.
8. L. Orth, Inaug. Dissert., Heidelberg, 1890. "Ueber die Sarkome des Darms, Mesenteriums u. Retro-peritonealen Raums."	F. 34 yrs.	Lympho-sarcoma (alveolar)	Transverse colon.	3 months.	Lymphatic glands of mesentery.	Resection (Czerny).	Had ovaries removed for sarcoma five years before; living and well three years later.
9. R. v. Baracz, Arch. f. klin. Chirurg., 1891, xlii. p. 501.	M. 8 yrs.	Round-cell sarcoma.	Cæcum; tumor formed apex of ileo cæcal intussusception.	13 weeks.	A nodule in neighborh'd of ileo-cæcal valve.	Lumbar incision; resection of tumor and intussusception.	Diagnosis: malignant growth in a movable left kidney or in descending colon. Death from shock second day.
10. E. Becker, Deutsche Zeit. f. Chirurgie, 1894, xxxix.	M. 35 yrs.	Round-cell sarcoma.	Cæcum.	5 weeks before operation; lived 47 dys. after operation.	Not stated.	Resection.	Recovery from operation; death from recurrence.

Reporter.	Sex and age.	Variety.	Location	Duration.	Metastasis.	Operation.	Remarks.
11. H. R. Wharton, International Clinics, 1894. 4th series, ii. p. 199.	F. under 2 yrs.	Round-cell.	Cæcum.	Sick several weeks before admission; still alive several months later.	To orbit.	Exploratory; portion removed for examination.	Symptoms simulated appendicitis before operation; when reported tumor was growing and orbital sarcoma had developed.
12. H. R. Wharton, personal communication.	M. 21 yrs.	Round. cell.	Cæcum.	About two months.	To peritoneum.	Exploratory.	Resembled acute appendicitis at onset; died of cachexia about six weeks after operation.
13. R. Abbe, Annals of Surgery, 1895, xxi. p. 592.	M. 6 yrs.	Not stated; no microscopical examinat'n	Cæcum and ascending colon.	Symptoms for 1 week.	Mesenteric glands.	Resection.	Death from shock 36 hrs. after operation.
14. C. Levi, Bull. de la Soc. Anat. d. Paris, 1895, lxx. p. 68.	F. 38 yrs.	Round-cell.	Sigmoid flexure.	2 months.	None.	Excision (Tuffier); formation of artificial anus; true nature not diag. until removed.	Death 2 days after operation; fatty liver, weighing 3200 grammes.
15. F. Smoler, Prager Med. Woch., 1898, xxiii. p. 145.	M. 27 yrs.	Lympho-sarcoma.	Cæcum, ileum, and ascending colon.	Not stated.	Mesenteric glands.	Resection.	Death 2 days after operation, from septic peritonitis.
16 F. Smoler. loc. cit.	M. 19 yrs.	Lympho-sarcoma.	Cæcum and ileum	Not stated.	Peritoneum.	None.	
17. Blauel. Virch. Archiv, 1900, clxii. p. 487.	M. 33 yrs.	Round-cell.	Cæcum and ascending colon.	18 months.	Retroperitoneal and portal lymph glands.	Tum. punctured, later incised and drained; opening communicated thro' tum. cavity with bowel	Colon bacillus found in fluid evacuated. Death from exhaustion 4 days after opening.
18. Blauel, loc. cit.	F. 66 yrs.	Round-cell.	Cæcum.	7 months.	Mesenteric and retroperitoneal glands and liver.	Exploratory.	Died 25 days after operation; anastomosis between tumor cavity and two loops of small intestine resembled tuberculous condit'n.
19. T. Fisher, Bristol Med.-Chir. Journ., 1901, p. 29.	M. 28 yrs.	Lympho-sarcoma.	Cæcum and ileum.	3½ weeks.	Few small subperitoneal growths in mesentery.	None.	Purpura hemorrhagica; infection by bacillus probably B. aërogenes capsulatus.
20. C. Van Zwalenberg, Journ. Amer. Med. Assoc., March 9, 1901.	M. 5 yrs.	Lympho-sarcoma.	Cæcum; partial intussusception of ileum and vermiform into cæcum	5 months.	Mesenteric glands and ileum.	Resection.	Recov.; 6 mo. later was well; mother and paternal grandmother died of sarcoma.
21. Hofmokl, quoted by Van Zwalenberg.	F. 24 yrs.	Lympho-sarcoma (adeno-(sarcoma)	Cæcum.	Not stated.	Not stated.	Resection.	Recovery.
22. Jopson and White.	M. 4 yrs.	Round-cell.	Cæcum and ascending colon.	Probably nine or ten months.	Mesenteric glands, peritoneum, kidney, spleen, and liver.	None.	Liver large and fatty, with some tumor infiltration.

An operation attended with a mortality of 50 per cent. is a formidable one, and not to be lightly recommended; but when we consider the brief duration of life without it—about five and one-half months being the average in the cases not operated upon—and the fairly good results in cases which survive resection, the operation, it would appear, should be advised when thorough removal seems feasible or likely. The section of the bowel should be made through a healthy area, well to each side of the microscopically diseased portion, in order to get beyond the infiltrating cells spreading, sheet-like, in the submucosa and subserosa. When, on opening the peritoneal cavity, the lymphatics and peritoneum are found extensively involved, and the liver and kidneys large on palpation—and, perhaps, nodular—simple closure of the wound will be all that can be done unless obstruction be present, when the formation of an artificial anus above the stenosed area would be indicated.

Van Zwalenberg,[10] in a study of 15 cases of resection of the bowel in various regions for sarcoma, found 9 recoveries and 6 deaths. Children stood operation very well. Of 5 cases under ten years of age only 1 died.

In preparing our list of cases we have excluded those reported by Perceval,[19] W. S. Thorne,[20] and Djemil-Pacha.[21] In Perceval's case the nature of the tumor is too uncertain to classify it; in Thorne's, from the histological examination, the tumor was probably a carcinoma, and in Djemil-Pacha's case, one of sarcoma of the great omentum and transverse colon, the tumor was probably primary in the omentum.

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ON THE NATURE OF THE DISEASE KNOWN AS ERYTHEMA INDURATUM SCROFULOSORUM.[1]

By Arthur Whitfield, M.D., M.R.C.P. Lond.,
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The disease first described by Bazin under the name of erythema induratum scrofulosorum has given rise to a great deal of discussion on the question of its pathology, and the doubts about its nature do not yet seem to be thoroughly dispelled. The report of the following two cases has, therefore, seemed to me to be justified, in the hope that some light may be brought to bear on an important and at present obscure train of symptoms. Before describing the cases upon which this paper is based I have deemed it advisable to sketch shortly the present position of the question. Bazin, the original describer, boldly attributed the disease to the scrofulous diathesis, and added the qualification scrofulosorum to his descriptive name of erythema induratum. The disease, according to him, generally affected young and well-nourished females, and this is a point which I think has, perhaps, too much dropped out of sight. Bazin's original description was rescued from oblivion by Colcott Fox and by Hutchinson, and in the renewed interest taken in the disease the question of the propriety of including it among the tubercular affections was raised.

As far as I know, Audry was the first to attempt to decide this point by excising a nodule from a patient suffering from the disease, examining it histologically, and also inoculating it into the guinea-pig.

His observations showed nothing at all resembling the tubercular structure in the excised portions, nor did his animals become infected with tubercle. The histology, according to him, was one of intense œdema with fatty degeneration, and he made no mention of the condition of the vessels of the part, confining himself to mentioning the rela-

[1] Read before the British Medical Association meeting at Cheltenham, August, 1901.

tion of the nodules to the sweat-glands. It is worthy of note that the age of his patient was thirty-four years. In the same year Leredde described a case occurring in a woman, aged thirty-six years, from whom nodules were also excised. No inoculation experiments were recorded, but a short histological note was given in which he drew attention to the fact that there was an extreme amount of phlebitis present, but no arteritis. He describes nothing at all resembling the usual tubercular structure, but winds up by assigning the disease to the class now known as the tuberculides.

Thibierge and Ravant then described three cases occurring in females, aged twenty-two, twenty-four, and fifteen years, respectively. In all three of these cases the histological appearances were those of tubercle, and in the last two inoculation of tissue into guinea-pigs was performed. The animal inoculated from Case II. (aged twenty-four years) was useless, owing to the fact that it was lost a few days later. Inoculation from the third case gave rise to typical tubercle of the animal, but it may be noted that the patient suffered from undoubted tubercle in one hand.

Since that time Dr. Colcott Fox has had an inoculation made from one of his cases, with a positive result, and other observers have published cases with histological reports showing evidence on both sides of the question. It should be mentioned that the age of the patient from whom Dr. Colcott Fox obtained his material was thirty-five years.

Within the last year it has been my good fortune to be able to observe somewhat closely, in the wards of King's College Hospital, two cases which illustrate two types of the disease, or, rather, I should prefer to say, indicate that two diseases are at present confused under the same name.

Case I.—A. M. C., aged fourteen years, was admitted into the wards for ulcers of the legs. The patient is one of thirteen children, eight of whom died in infancy of convulsions. The mother has had three miscarriages, but there is no history of syphilis or tuberculosis in the family.

The patient herself has always been healthy, and has never suffered from chilblains. About a month ago she first noticed some small red lumps on the backs of her legs. These were slightly painful and enlarged slowly, finally breaking down into the ulcers that are now seen. Soon after the appearance of the lumps on the backs of the legs she noticed a similar affection on the dorsa of both feet, close to the roots of the little toes. The patient is a healthy looking, well-grown girl, with rather high color and extremely clear complexion. There are no signs of past syphilis, nor, indeed, of any disease at all, past or present, with the exception of that for which she sought relief.

On the backs of the legs there are present several ulcers of varying size and shape (seven on the left leg and four on the right—see Fig. 1). These ulcers have deeply cut, clean margins, and the bases are covered with somewhat hemorrhagic sloughs. On palpation the ulcers are found to be formed by the central breaking down of a large

infiltration, the latter being indicated by the dark area in the accompanying photograph. Besides these ulcerated infiltrations there are several small nodules which are scarcely visible, but are very well defined to the touch. They lie deep in the subcutaneous fat and are freely movable over the deeper tissues, but are adherent to the skin. On the dorsum of each foot, opposite the base of the little toe, there is a small ulcer resembling those on the back of the leg.

She was given thyroid colloid in half-grain doses three times a day, and the legs were dressed with yellow oxide of mercury ointment. After eleven days all the ulcers except that on the dorsum of the left foot had cleaned and showed signs of healing, but the nodules did not seem to have altered in any way. Twenty-six days after admission the nodules were rather smaller, but still very definite, and on this day a strip of skin containing two of the largest was excised for me under a general anæsthetic by my friend Mr. Cheatle. Part of this was used for histological examination, and the other part was divided and inoculated under the skin of two guinea-pigs.

FIG. 1.

The wound made in the leg healed by first intention, and in this way two of the most resistant nodes were removed; but the other parts of the skin were very slow in completely resolving, and it was not until forty-five days after admission that the patient left the hospital, completely cured.

The two guinea-pigs inoculated with the material from this case never showed any signs of tuberculosis, a temporary enlargement of glands which occurred within the first fortnight afterward subsiding. One of these animals was killed at the end of a month, and the site of inoculation, together with the glands in the neighborhood and the spleen, were all examined microscopically, but without showing any evidence of tuberculosis.

The histological appearances of the excised nodule will be described with those of the second case.

CASE II.—A. C., aged thirty-seven years, married, was admitted to King's College Hospital, complaining of the presence of painful swellings in the legs. Her family history showed that she had two brothers who suffered from chronic cough, one being under treatment at a hospital for consumption. Her one child is delicate, being subject to tonsillitis. Her own history shows a severe attack of acute rheumatism at the age of fifteen years, since when she has always suffered from

dyspnœa on slight exertion or excitement. She has always been of an extremely hysterical nature. The present trouble began nine weeks ago by the appearance of some lumps on the backs of the legs, and soon after a few came on the sides as well. They were very tender even on their first appearance, the pain being of a severe, aching character and diminishing when the feet were raised. The lumps grew in size, but did not increase in number, and she had to lie in bed because of the pain. The result of this was that the swelling went down somewhat, and she was well enough to come to the hospital.

Present Condition. The patient is a slight, pale woman, with a well-marked tendency to acroasphyxia, which she says is worse in the winter. No internal disease is to be detected, but the condition of the heart is, perhaps, not quite normal. The dulness extends superficially up to the upper border of the fourth rib, and the apex-beat is displaced slightly outward. The beat is forcible, but neither diffused nor undulating, and the sounds are loud, but no murmurs are to be detected.

Both legs below the knees are affected by the disease, which is manifest as a series of small nodes which are almost invisible to the eye, being of a faint bluish-pink color, but are obvious enough to the touch, giving the feeling of small tumors about the size of a large pea to a nut, with very indistinct margins. These tumors are deeply seated, but are adherent to the skin and movable over the subcutaneous tissue. On careful examination it can be made out that the nodes lie chiefly over the internal and external saphenous veins, but there are also several which do not seem to bear any relation, at all events, to the larger veins of the legs. Immediately above the internal malleolus on the right leg there is a well-marked infiltration, about one and a half inches in diameter, which appears to be made up by the fusion of several of the smaller nodes, and over which the skin is distinctly reddened, shiny, and œdematous—in fact, looking as if it would readily break down to form an ulcer.

The patient complains of very great pain in the nodes, especially when they are handled or she stands up; in the latter case there seems to be some slight increase of size in the nodes. The contrast between the severe pain in this case with the slight symptoms in Case I. is very marked.

The treatment adopted was simply to raise the legs and wrap them up in cotton-wool. A few days after admission two nodules were excised deeply, the whole of the tissues down to the fascia being included. One of these was used for histological purposes and the other was divided and inoculated by Mr. Cheatle under the skin in two guinea-pigs.

At the end of a week after admission almost all of the nodules had entirely disappeared, and the large area of infiltration had completely subsided, leaving only a discolored patch. The patient only remained in the hospital for twenty-three days, and would not have been so long if I had not wanted to give her a prolonged rest, in order, if possible, to prevent recurrence of the condition. As regards the experimental inoculation, the animals both remained quite well and never developed any suspicious symptoms at all.

It will, I think, be best to describe the anatomy of the two cases together, for the purposes of comparison.

In Case I. the chief situation of the nodule is in the deeper part of the subcutaneous tissue. The infiltration consists of masses of small

round-cells distributed everywhere between the fat-cells, associated with a considerable overgrowth of connective tissue. In this connective tissue, which is œdematous, there are occasional foci which bear a strong microscopical resemblace to tubercular nodules, consisting of masses of epithelioid cells and some giant cells. (See Fig. 2). The small veins in this case show in many instances complete obliteration, with endothelial cells, so much so that only on careful examination can they be recognized as veins at all. The arteries show this change to a much slighter degree, if at all, and the sweat-glands, which have been described as being severely affected in some cases, appear almost normal here.

Sections stained by the Ziehl-Neelsen and other methods failed to reveal the presence of any organism.

FIG. 2. FIG. 3.

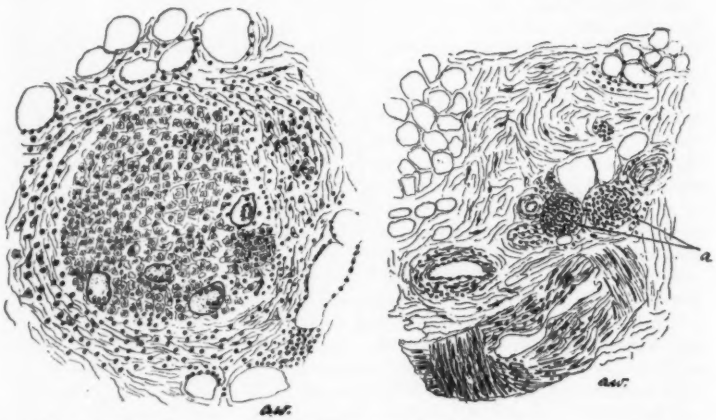

Fig. 2.—The granuloma suggestive of tubercle from nodule in Case I.
Fig. 3.—Phlebitis of venules (*a*) forming the only pathological change in Case II. No granuloma visible anywhere.

In Case II. the chief situation of the changes as regards depth is exactly similar to that in Case I. On microscopical examination, however, one sees at once that the infiltration is much less wide-spread than in the first case, being limited entirely to the neighborhood of a vein and its minute branches. Although numerous sections were examined, there was nothing at all resembling tubercle, or, indeed, any granuloma found in any section. The changes seemed to consist of a chronic inflammatory condition of the walls of the smaller veins, affecting chiefly the media and adventitia by infiltration with small round-cells (see Fig. 3, *a*) rather than by proliferation of the endothelial cells of the intima, as in Case I. No micro-organisms were found in any of the sections after special staining. We have here two cases of somewhat similar

history and appearance, except that the second seemed to be associated with more pain, especially on standing, and had not gone on to ulceration.

There are, however, marked differences to be noted, and in the first place I would draw your attention to the fact that in those cases in which histological examination has revealed an anatomy resembling that of tubercle the patients have been young, with the exception of Dr. Colcott Fox's case, in which the age was thirty-five years. In Bazin's description the age is not actually mentioned in years, but he describes it as occurring in young girls. In my first case the age was fourteen years, and the anatomy was that of tubercle. It is true that the inoculation test failed, but there may be two explanations of this. In the first place Dr. Fox has pointed out that these lesions are very rarely if ever progressive, in which case it may be that they are caused in some cases by dead tubercle bacilli, since we know that embolism by dead tubercle bacilli will cause the production of non-progressive tubercles. This, of course, is not always the case, or positive inocula tions would never result. Secondly, there was a long period of rest and treatment before the nodules were excised. This is a lasting regret to me, but was unavoidable, since Mr. Cheatle's license had been allowed to lapse while he was away in South Africa, and we had to wait some time after application before he obtained a new one.

In the light of this point I think we may assume that the first case was in all probability tubercular. The second was certainly not so. No time was lost in obtaining the tissue, and yet there was no result from our inoculations, nor was there the slightest resemblance to tuberculosis in the anatomy of the lesion. The age of this patient is similar to that of Audry's and to Leredde's; and although Leredde considered his case was one of tubercle, he made no inoculations, nor do his own published descriptions suggest tubercle to the reader. The remarkable way in which these lesions in my second patient cleared up on rest alone was totally different from what occurred under similar circumstances in the first case, where the non-ulcerated nodules took weeks to clear up, and also tends to show that we had two different forms of the disease under observation.

The conclusions I would draw, then, are that there are at least two forms of disease to which the name of Bazin's disease has been given. One of these diseases is essentially indolent, chiefly affects adolescent girls, and is in some cases undoubtedly caused by the living tubercle bacillus. The other affects women of middle age almost if not quite entirely—usually those who suffer from some form of cardiac weakness—is more acute, more amenable to treatment by rest in bed, and has nothing to do with the tuberculous process.

A case allied to if not identical with this second condition has recently been described by Philippson under the name of phlebitis nodularis

necrotisans. His patient was, however, a girl who suffered with the disease from the age of eighteen to twenty years, and apparently the affection was much more severe than was the case in my second patient. The histological findings were identical in the two cases.

Since this paper was completed a further report of two cases by Professor Mantegazza has appeared. Both his cases were young girls (aged twelve and fifteen years, respectively), and the histology showed a definite granuloma resembling tuberculosis, with great changes in the arteries and veins.

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A CASE OF LUPUS ERYTHEMATOSUS CURED BY THE X-RAY.

By Richard F. Woods, A.M., M.D.,
of Philadelphia.

I wish briefly to report the following case of lupus erythematosus, which I think is of considerable interest from the fact that it was entirely cured by the application of the X-rays.

I think the rapidity of cure was quite remarkable. The lesion, which had been steadily growing for four years, was entirely eradicated in five exposures to the rays, each treatment lasting ten minutes.

The history of the case is as follows:

M. D., aged thirty-two years. No tubercular history. Four years ago the patient noticed a small pimple on her face, which she constantly irritated with her finger. It steadily increased in size, giving her some pain, and bleeding quite freely at times, and especially worried her in cold weather.

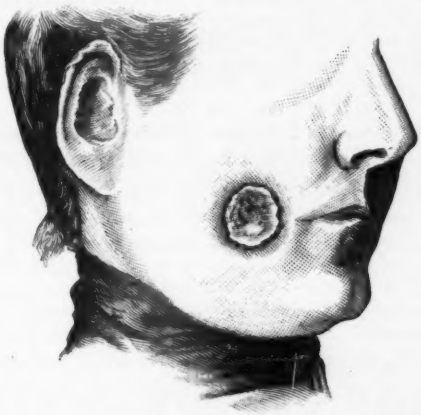
She had been attending clinics in several hospitals in this city for the past two years, and had applied various ointments and had taken medicine internally without success.

She presented herself at my office on July 12, 1901. The growth at that time was about the size of a half-dollar, situated slightly above the angle of the mouth (Fig. 1, which was made at that time). It was of the discoid form, slightly raised, and of a bright reddish color. The centre of the growth was partially covered with scales. The erythematous border was sharply bounded by the skin, and was firm to the touch. There was some alteration in the mouths of the sebaceous

glands; the follicular orifices were enlarged, gaping, and filled with hardened sebaceous matter.

I suggested the X-rays, and she was given her first treatment two days later at the Pennsylvania Hospital, with the assistance of Dr.

FIG. 1.



Mitchell. The face was covered with a sheet of lead from which a small V-shaped piece had been removed to expose the growth. This was thought necessary to protect the face from burning. The ordinary Ruhmkorff coil was used, charged from the electric-lighting current, and

FIG. 2.

had the strength of about eleven volts. The current was turned on for ten minutes, and the distance between the face and the tube was seven inches. The treatments were continued twice a week.

When the patient returned for the second exposure the improvement was remarkable. The entire character of the growth had changed. There was a diminution in the color and the prominence of the edges.

The erythematous halo was fading away, and the raised margins had become level with the sound skin, while the central portion was becoming depressed and atrophic. The improvement continued rapidly, and when the patient returned for the sixth treatment it was found unnecessary to continue. The area of ulceration had entirely healed, and was covered with a healthy cicatrix. The photograph (Fig. 2) was taken a few days after the last treatment, and shows the appearance of the scar over the original site of the lupus. Through trouble with the camera we were unable to get a good picture of the patient before the treatment was started, but the drawing shows fairly well the size and condition of the growth.

The rapidity of cure in this case of tubercular disease of the skin (although some authorities doubt the tubercular nature of lupus erythematosus) is very satisfactory, and, with the other cases that have been reported, would warrant a broadening of the field of experimentation with the X-rays in other tubercular affections.

A CASE OF LYMPHATIC LEUKÆMIA, APPARENTLY DEVELOPING OUT OF HODGKIN'S DISEASE, ACCOMPANIED BY LEUKÆMIC LESIONS AND PIGMENTATION OF THE SKIN, CULMINATING IN STREPTOCOCCUS INFECTION.

By Grover William Wende, M.D.,
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(*From the Pathological Laboratory of the University of Buffalo.*)

Summary of Chief Points. *Hodgkin's disease, first shown by physical and blood examinations; lymphomata in the skin, the first evidence of the disease developing where lymphoid tissue does not normally exist; subcutaneous and internal hemorrhages; spontaneous increase and reduction of tumors, characteristic of Hodgkin's disease; suddenly developing clinical and blood changes, showing transition from pseudoleukæmia into true lymphatic leukæmia; terminal tonsillar infection, resulting in general sepsis, with disappearance of blood picture of lymphatic leukæmia; subsidence of lymph nodes and spleen during infection. Personal History.* The patient, J. B., aged twenty-six years, first seen on April 26, 1900. His father died at the age of fifty-eight, presumably of some malignant disease of the bladder, and his mother at the age of forty-eight of inflammation of the bowels. His four brothers and one sister survive him, and have always had excellent health. To his knowledge none of his relatives, near or remote, ever suffered from his particular complaint or from any cutaneous affections. His habits were never vicious, he was never affected by syphilis.

Previous History. No assignable cause for patient's condition. It began with a slight induration of the epidermis and its underlying structure over the left temple, midway between the eyebrows and the margin of the scalp, first accidentally observed by the patient December

1, 1899, five months before he came under my notice. At first this increased very slowly, afterward more rapidly. For two months it was confined to the initiatory seat, during which period it attained the size of a twenty-five-cent piece. The patient then consulted a physician, who pronounced the lesion syphilitic gumma, and prescribed accordingly; as, however, under this treatment, there was no improvement, electrolysis was resorted to, which only served to stimulate the growth. A circumscribed hardness now became apparent in the centre of the cheek on the same side, though deeper in the cutis, and without change of coloring in the integument, the parts losing their suppleness. The patient experienced a hide-bound sensation whenever the jaw was moved. This manifestation, beginning on the left side, was irregular in outline, with a mean diameter of about five centimetres. The lymphatic nodes were enlarged from the moment of its inception, first in front of the ear, and subsequently involving the post-auricular, which became intensely painful. Later the inferior maxillary, and finally the cervical were affected, the latter attaining a considerable size. The accompanying pain and soreness were of short duration.

On February 20th a number of superficial, irregular, yellowish-gray spots were detected over the region of the sternum, while at the same time a slight infiltration manifested itself in close proximity to the left nipple, which increased with amazing rapidity. Other areas appeared correspondingly distributed over each side of the chest and back. These discolorations were of varying sizes and shapes, but their borders were well defined. As a sequence of this general dissemination, there ensued an enlargement of the lymphatic glands of the axillæ, which were more or less tender upon palpation. Apart from the slight tenderness of the glands, the patient suffered no inconvenience. Appetite good, habitually slept well, and continued to apply himself to heavy manual labor.

Examination of the Patient. Upon his arrival at Buffalo he was examined by Dr. Charles G. Stockton, who pronounced his organs healthy. No enlargement of the spleen discoverable. The examination of the blood, which was made by Dr. Albert E. Woehnert, showed at this stage no marked alterations, being as follows:

Erythrocytes	5,128,000
Leucocytes	4,000
Hæmoglobin	88 per ct.
Lymphocytes (small)	27 "
Lymphocytes (large and transitional)	4 "
Polymorphonuclear neutrophiles	68 "
Eosinophiles	1 "

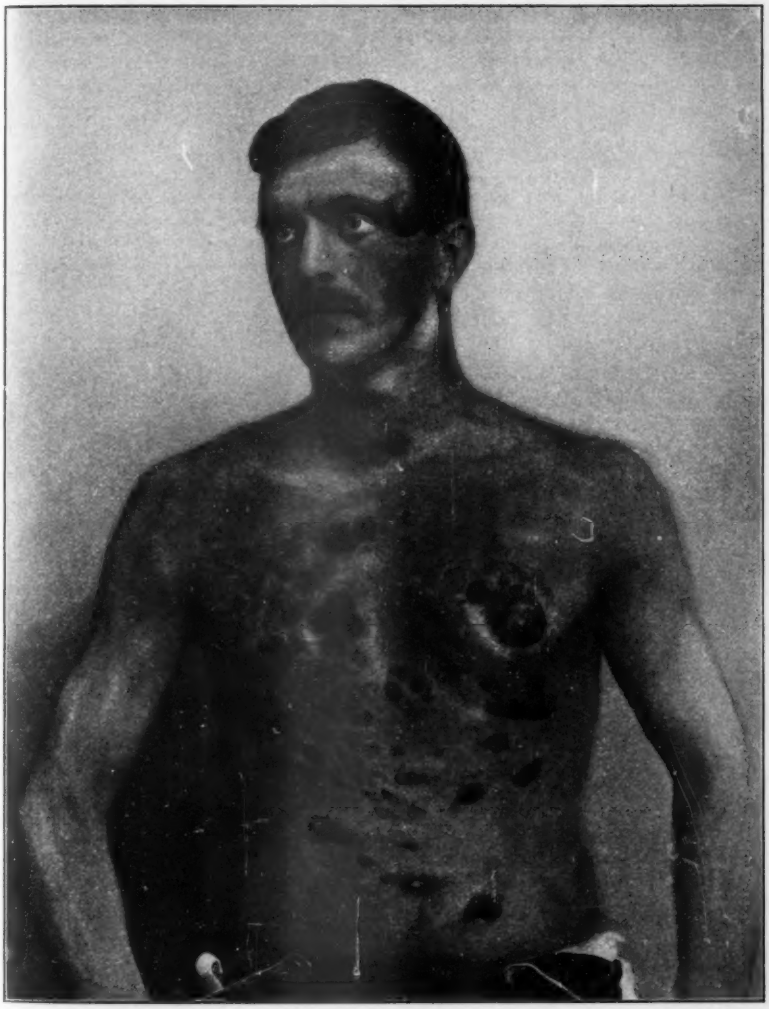
In counting the leucocytes, microcytes and microblasts were occasionally discovered. The red corpuscles were apparently somewhat irregular and pale. The urine had an acid reaction; specific gravity 1020; urea and uric acid slightly augmented; sugar and albumin absent; no casts or leucocytes, and but few crystals of uric acid.

The illustration of the case here presented was taken but a few days prior to the clinical record. It fairly demonstrates the topography of the multiple formations. The general tint of the skin was ruddy, as though sun-burned, while the areas upon the chest were as brown as burnt sienna—faithfully reproduced by the artist. The initial lesion, situated on the left side of the face, occupying the upper part of the temporal, the lower part of the frontal, and the exterior half of the

supraorbital regions, formed an oval protuberance, with its long diameter extending from a point beneath the eyebrow to a point beyond the hairy portion of the scalp, where the individual hairs were raised like the spines of a porcupine. Its greatest length was nine and its greatest width ten centimetres. It was non-adherent beneath, sliding readily with the skin; the border was well defined and its dusky hue was without transition or the slightest erythematous tint, which was in keen contrast with the normal epidermis. The whole growth was hard and elevated above the contiguous skin. The entire surface was not only distinguished by its cyanotic tinge, but by numerous vibices, some in streaks, others cruciform. In the profile of the right eyebrow were two definite nodules, about the size of a pea, freely movable and in keeping with the normal color of the skin.

The centre of the left cheek bore a deep-seated infiltration of three and one-half to four centimetres in diameter, presenting an unaltered hue, except at the centre, which bore a tawny shade. The face lesions were four in number; the scalp was in nowise involved. Seated upon the body over the sternum were two lesions, each measuring ten to twelve millimetres. The infiltration of the upper one was invariably thin and invested with a yellow pigmentation. In the lower one the infiltration was more pronounced and its circumference more definite; it presented a bluish hue and showed a number of minute red spots, more or less irregular in contour. In the left inframammary region, approaching the mesial line, were two spots about the size of a filbert, covered with normal skin, and deeply seated. On the left breast a large patch had formed around the nipple, similar to that situated on the temple. The areola of the nipple had disappeared. The surface of this patch was greatly elevated, and was oval in outline, with a long diameter of ten centimetres, quite hard, easily displaced, and more or less reticulated except at the centre and lower edge, which were shrouded with vibices. Situated on the lower portion of the abdomen were seven distinct and deeply-seated nodules, varying in size from a pea to a hickory-nut. In four the skin color was unaltered. The largest, and the one first developed, was of a cyanotic character, covered with a brown pigmentation. The peculiar features of all the lesions are not visible in the photographic reproduction, owing to the fact that they were so deeply embedded, without apparent change in the epidermis. The whole chest and back were mottled, showing numerous, well-defined pigmentary stains, and variegated blotches of different shapes and sizes. There must have been forty or more upon the anterior surface of the trunk, and nearly as many upon the posterior. These differed in size from a pea to a twenty-five-cent piece, and in some localities coalesced. They usually presented a circular form. This was especially true of the large ones—the smaller were frequently misshapen, sometimes grotesque. The prevailing color was a chocolate yellow. Barring the discolorations, the epidermis was seemingly normal. There was a decided hypertrophy of all the prominent superficial glands. The first to show any appreciable increase were two anterior to the left ear, just above the parotid. The post-auricular of the left side was the size of a hazelnut, and very sensitive. The post-cervical soon assumed the dimensions of a black walnut, but was not specially sensitive. The entire cervical group and the submaxillary glands were so enlarged as to be palpable. The glands of the trunk, with the exception of the epitrochlear

and inguinal, were more or less involved and possessed uniform sensitiveness. The left axillary was the largest, the size of a walnut. On the whole, this hypertrophy of adenoid tissue was greater about the



head and neck than upon the trunk, and less upon the lower extremity. The magnitude of the glands of the right side was not more than one-half that of those on the left side. There was no evidence of adhesions, the glands being readily separable. As a rule, the smaller were much

harder than the larger ones. This briefly describes the condition of the patient when first seen.

Clinical History. Three days later there was a marked change in the primary lesion, the dimensions having increased four millimetres. This rapidity in the development seemed to demand its immediate removal, especially as its exact nature was not known. The operation was performed on the following day under cocaine infiltration, by making an incision around the growth. The edges were brought together by sutures, the wound healing quickly and bearing a fair linear cicatrix. The material of this growth, together with that of growths on the other portions of the body, was used for microscopical examination, to be described later. It will be sufficient at present to say that the growth was of a lymphomatous character. The patient was afterward placed under the hypodermic administration of arsenic. The subcutaneous injections began with six drops of Fowler's solution, increased two drops weekly, daily administered, and within three weeks all the manifestations showed improvement. New lesions ceased to form, the old ones manifested less infiltration, and all assumed a similar discoloration, which in the course of four weeks changed to a dark brown, and finally began to be absorbed at the periphery of the various smaller lesions. At the end of six weeks the larger infiltrated patches had practically cleared up, and the tumors disappeared, as well as the enlarged blood-vessels and cyanotic tinge. The skin resumed its natural aspect, except in the matter of pigmentation. During this period the improvement was progressive, but on June 4th there was an unexpected return of the difficulty, the glands taking on their former proportions. For the abruptness of this return there was no assignable cause. The new condition was, however, brief, for, after the lapse of thirty-six hours, the tenderness subsided and the hypertrophy of the glands gradually began to decrease. The general condition of the patient during the paroxysm was good; his appetite was excellent; there was no rise of temperature; the secretions were healthy, and the excretions regular; there was no change in the organs; the urine was normal. He was now receiving twelve drops of Fowler's solution hypodermically administered each day. Everything went well until July 1st, when it was noticed that the condition was undergoing a change. The skin assumed a shade of light bronze, especially pronounced on the back, the chest, and hypogastrium, and three growths suddenly appeared, one on the scalp and two on the back.

On July 7th it became necessary for him to return home. However, he continued the treatment during his absence, and returned on July 10th, when another lymphatic enlargement ensued, involving the inguinal glands on the right side, which were greatly increased and seemed hard to the touch. The largest of the cervical chain, which was apparently encapsulated, was now extirpated. Its contents were quite soft, appearing very much as if they had undergone mucoid degeneration. At the same time a change took place in the general condition of the patient. His physique was less robust and his gait less sprightly; he seemed drowsy, stupid, and weak; the mucous membranes began to show paleness; the enlarged glands manifested an appreciable increase, as did many others not previously affected. Those under the ramus of the lower maxillary gained the most, causing a pressure which provoked a cough. Expectoration was increased.

Petechiæ appeared for the first time upon the shoulders. Fresh hemorrhages, which encroached upon the back and chest, occurred daily, gradually extending down the arms. These hemorrhages developed slowly and were limited to the localities mentioned. The greatest number of confluent lesions was formed upon the chest. In certain regions they were thick and circumscribed, or of a linear configuration, and at every new outbreak were conspicuous, red, and bright. In size they varied from a pin's head to a pea, well defined, and uninfluenced by pressure. During the ten days following their original manifestation both sides were profusely studded with them. They were also present upon the eyelids and conjunctivæ; the ophthalmoscope revealed their existence on the retina of each eye and showed that the bloodvessels were dilated and tortuous. Pigmentation was marked and rapidly increased. This was especially pronounced over the hemorrhagic area. In fact, the entire surface showed both a rapid transformation and a decided increase in color, the earlier idiopathic pigmentary spots becoming so dark as to resemble nitrate of silver stains. Many of the spots on the chest coalesced, and, the skin becoming generally pale, the contrast was naturally great. There were no evidences of pigmentation in the mouth.

An erythematous discoloration was also in evidence, especially well defined upon the arms. This readily disappeared upon pressure. From these prominent changes in the skin it was naturally concluded that the blood might demonstrate equally definite alterations. The mucous membranes and the finger-tips exhibited a peculiar pallor. Nothing unusual was found upon physical examination, with the exception of a systolic murmur. Pulsation of the bloodvessels of the neck was clearly visible, those of the temporal region being especially conspicuous. A provisional diagnosis of lymphatic leukæmia was made, and the blood examination resulted as follows:

Hæmoglobin	40 per ct.
Specific gravity	1,043
Red cells	1,936,000
Macrocytes present in moderate numbers.	
Microcytes the same.	
A few poikilocytes.	
Red cells somewhat pale.	
102 normoblasts to the cubic millimetre.	
White cells	34,000
Small lymphocytes	95.5 per ct.
Large lymphocytes	0.7 "
Eosinophiles	0 4 "
Polymorphonuclear neutrophiles	3.4 "

The lymphocytes varied somewhat in size, the majority, while conforming to the normal contour, were of greater dimensions than those of the ordinary cell.

The physical examination now made revealed no appreciable rise in temperature, yet the pulse recorded 110 beats per minute. A slight fulness in the left epigastric region was noticeable, and the spleen was somewhat enlarged, the estimated perpendicular diameter being about ten centimetres above normal. The organ was apparently smooth; both deep pressure and percussion over the sternum proved painless.

July 18*th*. The glands about the neck, having rapidly augmented, became distinctly individualized, and resembled a festoon which swept

around the inferior maxillary bone and extended from ear to ear. With the exception of a few that were small the knots attained the dimensions of a hen's egg. Owing to their location and extraordinary proportion they were tender and sensitive, occasioning great pain and increased flow of saliva. The gums were blue, swollen, and spongy, with a tendency to bleed upon the slightest provocation. The teeth were covered with sordes, and the lips were dry. In connection with this peculiar enlargement, soreness of the throat set in, which, in the form of a hypertrophy, was mostly limited to the tonsils. These within a week grew to such an extent as to impinge upon each other. The thyroid gland was not apparently involved. The patient became easily fatigued, his appetite was impaired, and he could not sleep. There was some dyspnœa, with a frequent cough, due, in all probability, to the enlargement of the tonsils. An analysis of the urine showed it to be about normal in color; acid reaction; specific gravity 1020; no albumin; no sugar; yellowish-white sediment; no casts; uric acid crystals; uric acid 0.784 grammes.

19*th*. The patient was now confined to the house. Following a severe paroxysm of coughing the purpuric eruptions appeared for the first time upon the cheeks, neck, and back; heretofore they were almost invariably found only upon the chest. Morphine was administered with little or no effect. The gums continued to swell until they nearly covered the teeth, ulcerations were discovered surrounding the bicuspids, and the bronzing of the skin became more and more pronounced.

20*th*. The patient appeared weaker and more languid, was still sleepless, and complained bitterly of pain about the region of the neck. His temperature was 99.5°, pulse 120, with a slight outbreak of purpura.

21*st*. Condition unchanged. Examination of the blood showed that microscopically it was pale and thin; coagulation took place slowly and irregularly.

Red cells	1,776,000
Microcytosis and macrocytosis fairly marked.	
Poikilocytes in fairly large numbers.	
Nucleated cells 225 to cubic millimetre.	
A number of polychromophiles.	
White cells	45,000
Small lymphocytes	95.3 per ct.
Large lymphocytes	1.6 "
Polymorphonuclear neutrophiles	2.3 "
Eosinophiles	0.6 "
Myelocytes	0.2 "

Temperature 99.8°, pulse 124. The lower lip became suddenly œdematous, increasing to three times its natural size.

22*d*. Nausea and vomiting, with an outbreak of purpura in close proximity to the hairy scalp. No new spots on the body. Deafness of the left ear, accompanied by roaring and ringing, the result of hemorrhages in the meatus and on the tympanum.

23*d*. The hemorrhagic eruptions gave evidence of becoming paler, and after the lapse of three days had almost entirely vanished; restlessness increased.

26*th*. The pain in the throat was augmented, especially during deglutition, the right tonsil having become ulcerated. The ulcer was

as large as a five-cent piece, well defined with sloughing edges. The œdema of the lips had sensibly diminished. The general tumefaction of the lymph nodes that was so characteristically marked had begun to subside. This was especially true of the glands beneath the inferior maxillæ. Temperature 100°, pulse 140.

27*th*. At one o'clock in the morning the patient was subjected to severe retching and vomiting. On the following morning the entire cutaneous surface was much reddened, with subcutaneous hemorrhages, affecting even the loose cellular tissue of the eyelids. Patient swallowed with difficulty; external pressure over the tonsils caused great pain. New symptoms appeared in the form of diarrhœa and dark-colored urine. The abnormal color in the urine disappeared the same day, but the stools were frequent and copious and continued until the day following. The glands had materially decreased. Urine passed, 2040 grammes; specific gravity, 1021; uric acid, 1.44; slight amount of albumin, no sugar.

28*th*. Symptoms more intensified; condition weaker and much depressed; the ulcerated tonsil extremely sensitive. Patient vomited at noon; retching not marked; no purpura followed. Temperature 104°, pulse 140. The urinalysis of specimen passed in twenty-three hours showed the amount to be 2940 grammes; specific gravity, 1023; color high; aromatic, but not disagreeable; reaction acid; urophen and indican increased; urea, 39.403; uric acid, 2.64; sulphates, 9.364; phosphates, 3.470; no bile pigment; no sugar; no albumin; sediment consisted of squamous epithelial cells, probably of urethral origin; red cells not detected; leucocytes plentiful; a few hyaline casts; sediment thickly covered with uric acid crystals. Bacteriological examination by swab from throat revealed streptococci and various other micro-organisms.

29*th*, 8 A.M. No appreciable change in patient's condition except that he was more depressed. Pulse 150, temperature 104°. At 5 P.M., pulse 160, temperature 105°, pulsation of the carotid distinctly visible; no desire for food; extremely weak; slough on tonsil mostly gone; its edges and base were bright red, while the adjacent mucous membrane was pale. Patient complained bitterly of lancinating pain on the inner side of instep. Spleen showed no increase, as before. Percussion over the sternum no longer painful. The amount of urine voided for the past twenty-four hours was 3240 grammes; specific gravity, 1021; uric acid, 4.94 grammes. At midnight, temperature 106°, pulse 160, respirations 50. Epistaxis discharge usually pale; evidence of coagulation where it came in contact with the towel. The bleeding ceased at 1 o'clock. Patient complained of pain over the plantar arch of both feet.

30*th*. In the morning patient not so restless, but much weaker; pulse 160, temperature 106°, respirations 40 to 50. In the evening he was decidedly weaker. Pulse 180 and very thready, temperature 107°. Sponge-baths continued as the only means of effecting any relief. Patient complained of discomfort in his right hand, which was more or less œdematous on dorsal surface; the same was true of the free border of the ribs on the right side of body. Mouth parched; bowels moved involuntarily; the lymph nodes could only be detected upon palpation. The blood examination twenty-four hours before death demonstrated that it was very thin and pale, with only a suggestion of coagulation.

For this purpose it was taken from lobe of left ear, and gave the following results:

Red corpuscles	803,000
Leucocytes	1,600
Specific gravity	1030
Hæmoglobin	30

Normocytes pale, microcytes moderate number.
Megalocytes large number.
Poikilocytes.
Schistocytes few.
Normoblasts none.
Blood-plates increased.

The following relation existed among the leucocytes:

Small lymphocytes	88 per ct.
Lymphocytes (transitional and large)	1 "
Polymorphonuclear neutrophiles	10 "
Eosinophiles	1 "

The last examination (incomplete) was made ten hours later, or just before death. This revealed a remarkable reduction of the leucocytes, namely, to 600 per cubic millimetre. At midnight the pulse was weak, rapid, thread-like, and intermittent; temperature approached 108°, respirations 38, and tubular in character; heart sounds almost inaudible; general condition greatly relaxed and very feeble. At 2 A.M. patient powerless and flaccid; temperature still elevated; at times slightly delirious, but when spoken to would answer intelligently; heart sounds inaudible; respirations gradually diminishing; easy death at 3.30 A.M.

Autopsy Notes. An autopsy was made eight hours after death by Dr. Herbert U. Williams. Body fairly well nourished; rigor mortis quite firm; pigmentation of the skin over the upper part of the chest and back and distributed irregularly, that upon the chest most marked, the patches being about the size of a dime. In other places they had coalesced and had formed irregular patches. The rest of the skin covering the body presented a dark-gray appearance. Petechiæ appeared upon the chest and were isolated near the pigmented border, although confluent toward the centre. There were three small tumors in the skin covering the abdomen. On section they showed a whitish appearance. Some superficial lymph nodes gave evidence of enlargement upon palpation. The cervical, supraclavicular, and inguinal were the most pronounced, although manifesting a marked decrease in size compared with their dimensions before death. They were of firm consistency and freely movable. The subcutaneous adipose tissue was small in amount.

Thorax. The pleural cavity contiguous to the smaller bronchi was studded with lymph nodes about the size of a pea. The peribronchial lymph nodes varied in size from a pea to a chestnut. Upon repeated sections they looked like raw flesh, apparently the result of hemorrhages. No caseation was present. The thymus gland was patent.

Lungs. Lungs somewhat dry, pale, and presented nothing of interest except a few small superficial hemorrhages. The pleural cavities contained no fluid.

Heart. The pericardium contained about three ounces of perfectly clear serum; the parietal layer was covered with minute petechiæ. The lymphatics of the outer surface of the heart were dilated with lymph to the size of a straw. The right ventricle was distended, containing a pin clot and a small amount of blood. The left ventricle was contracted and nearly empty; its posterior wall had a petechial spot. The heart muscle was firm and pale, and the valves were all in a normal condition. The peritoneal cavity contained a small quantity of serum, but was otherwise apparently normal. The omentum was distinguished by enlarged lymph nodes which were about the size of a small pea. The retroperitoneal lymph nodes also showed definite enlargement.

Stomach. The various layers appeared normal, with the exception that throughout they were studded with enlarged lenticular follicles about the size of a French pea.

Intestines. The contents consisted of a semi-fluid, yellowish-white milk stool. Marked hypertrophy of the mesenteric lymph nodes was noticed. These varied in size from a pin-head to a pea. The solitary follicles were enlarged. Near the ileocæcal valves Peyer's patches showed thickening. The vermiform appendix was normal.

Spleen. Weight, one-half kilogramme. On section trabeculæ not distinct. Malpighian bodies large and well marked.

Liver. Weight, two and one-half kilogrammes. Smooth and pale in color. On section very coarsely mottled with varying shades of red, corresponding with areas comprising several lobules. A substance covered the diaphragmatic surface which looked like fibrin. The gall-bladder contained about 43 grammes of pale bile, otherwise normal.

Kidneys. The ureters were normal. The left kidney was large and soft, weighing about 175 grammes. The capsule was pale and covered with petechiæ, and was easily stripped off; cortex very broad, having a swollen and hemorrhagic appearance. The cortex was covered with peculiar pale-gray streaks. The right kidney was the same as the left. Suprarenal bodies normal.

Bone-marrow. The bone-marrow of the sternum was slightly paler than normal.

Bacteriological Examination. During life the following examinations were made: Portions of the tumor removed from the breast were placed in the peritoneal cavity of live dogs. The blood of the animals was examined at two different times without any change. Four months later the dogs were killed and the blood was found to be in a healthy state. No enlargement of the lymph nodes or other abnormal condition was noted.

Inoculations made from a tumor into various culture media unfortunately became contaminated. There was difficulty in getting cultures from the skin. Swabs on agar made from the ulcer of the throat three days before death, placed in the incubator and examined after seventy-two hours, showed various cocci and bacilli, among them numerous streptococci. Examination in sections of the tumors removed during life for bacteria, also of the blood for the parasites described by Lovett, were negative.

The following results were obtained at the post-mortem: Smears made from the various organs were all negative, with the exception of those made from the spleen, which showed the streptococcus only sparingly. Cultures, including anaërobic, were made on different media at

the post-mortem and placed in the incubator. The cultures on agar were made from the lung, blood of right and left ventricle, liver, kidney, bronchial lymph node, post-peritoneal lymph node, spleen and bone-marrow. Cultures from the bone-marrow and lymph nodes were also made on human blood serum. Streptococcus pyogenes were grown from all the organs. The staphylococcus pyogenes aureus was recovered from a bronchial lymph node along with the streptococcus. The post-peritoneal lymph nodes showed an organism resembling bacillus coli communis, and the kidney a large bacillus resembling the hay bacillus. These were regarded as post-mortem invasions without significance.

Histological Examination—Cutaneous Lesions. For the microscopical investigations made during life, in addition to the material obtained from the initial lesion on the forehead after its removal, fragments were also procured from the affected skin surrounding the left nipple, from the patch located over the sternum, and from another patch situated upon the abdomen. These were hardened in substances conventionally recognized for such purpose. The paraffin sections cut therefrom were stained by the usual method. These various sections showed microscopically that the tumor was essentially of a similar structure throughout. The fundamental tissues largely consisted of lymphoid cells. The growth proper principally occupied the structure of the cutis and subcutaneous tissue. Its greatest development took place in the vascular reticulum of the latter. The cellular infiltration was so great that it pressed apart the normal structures. The cells of the adipose tissue were widely separated by this diffuse process, and the layers much increased in thickness. In the superior partis vascularis of the corneum, at the periphery of this growth, there was comparatively no infiltration, while it was exuberant and characteristic about the inferior partis vascularis and around the bloodvessels adjacent to the subcutaneous tissue. The thickness of the infiltration varied considerably, reaching as high as 2 cm. These abnormal cells were usually round, except where the shape was modified by pressure. They consisted of a homogeneous protoplasmic rim which took acid aniline stains and a relatively large, round nucleus. Each nucleus seemed to possess a membrane, and a distinct round nucleolus occupied its centre. No mitoses were seen. The nuclei possessed a characteristic network, forming a rim with numerous dark points within it. Certain stellate cells, probably connective-tissue, were found, possessing large protoplasmic bodies and yellow pigment granules. These were not always present, although found in the majority of specimens of the true skin and subcutaneous tissue. The deep epithelial cells were generally pigmented. Isolated masses of pigment were found in all the growths investigated, and were especially prominent in the deep-seated parts. In looking over the sections that were stained with polychrome methylene-blue for mast cells they were seldom detected either among the cells of the tumor or connective tissue. Plasma cells were never discovered. The hæmatoxylin stain demonstrated the presence of numerous homogeneous bodies of irregular or roundish contours, with a well-defined nucleus, that were much smaller than the tumor cells. They took the dye deeply, were well outlined, and were most frequently encountered in the true skin, less often in the connective tissue, occasionally in the striped muscles, and but rarely in the centre of the tumor. In structure and appearance they closely resembled the parasites claimed by some writers to exist in

certain tumors as well as in connection with other affections. They might, however, have been the result of nuclear degeneration. Many sections showed small clusters of three to six in the reta cells, which had apparently lost their prickles, yet their nuclei were well stained. If these bodies were the products of degeneration those found in the muscle must have been derived from the muscle-cells, those in the connective tissue were probably disorganized nuclei, and those of the tumors may possibly be regarded as the result of nuclear degeneration.

The trabeculæ of the connective tissue in the new growths consisted of a reticulum of interlacing bands, or fibres, least conspicuous where the infiltration was intense. This reticulum contained the lymphoid cells, and the whole arrangement was suggestive of a lymphadenoma. Using Weigert's stain for elastic tissue, elastic fibres could easily be demonstrated in the neoplasm. Especially was this true in those portions of the growth where the process was most active. The elastic fibres were rather more numerous in the large tumors. They were most abundant about the sweat-glands and hair-follicles. In the walls of the bloodvessels, wherever there was any thickening, the elastic tissue was apparently augmented. The arteries in the tumor masses sometimes showed a thickening of their walls from an infiltration with lymphoid cells. This process was most marked where the tumors were the largest.

Early in the infiltration the integrity of the hair follicles and their appendages were well maintained, but later on they were surrounded by an accumulation of round cells, when their outer sheaths became infiltrated and finally obliterated the follicle, the hair alone remaining.

The alterations in the sudoriferous glands varied with the location. Those in contact with the tumor showed the reproductive nucleated cells within the lumen to be swollen, and at times completely filled the duct. Later they became so modified and disarranged as to be confused with the neoplastic cells. In those portions of the neoplasm most highly developed they were entirely obliterated.

The sebaceous glands seemed for a long time to resist the infiltration, but when completely surrounded and penetrated by the new-growth cells were apparently made to disappear.

Every layer of the skin was infiltrated, with the exception of the stratum corneum, this being somewhat modified by the pressure of the cellular infiltration from beneath, varying much in thickness, from a few cells in some localities to the normal condition in others. In no instance could the cells of which the tumor was composed be found to infiltrate the epidermis. There was no evidence of porokeratosis or desquamation. The changes noted were important, in that they were characteristic of the disease under consideration. The reta mucosum in certain places was much altered and varied so greatly as to force the conclusion that these changes were secondary to the tumor formation. The interpapillary processes were obliterated by pressure. Not a few of the prickle cells were more or less swollen by reason of œdema of the interepithelial spaces. Here and there in the rete were vacuoles. In these free spaces leucocytes could occasionally be seen. The stratum granulosum was conspicuous and consisted of two or more layers, while the stratum lucidum could only be recognized at certain points. The pigmented skin, which was free from tumor, showed a pronounced increase of thickness in the granular layer, which encroached upon the stratum mucosum. The pigment consisted of brownish-yellow or black

granules, uniformly deposited, which could likewise be detected in the ducts of the sudoriferous and sebaceous glands.

Internal Organs (Microscopical). On microscopical examination of the tissues of the various internal organs, which were placed in Zenker's fluid at the time of autopsy, the leukæmic infiltration was found to have affected all the organs. It was not, however, as pronounced as in the masses taken from the skin.

The bronchial lymph nodes showed a great increase of the cellular elements. The capsules were thin, and there was no evidence of inflammation around the nodes. Adjacent to these were many hemorrhages and numerous pigmented cells. Hemorrhages were discovered in the nodes, and there were numerous red blood-corpuscles among the lymphoid cells. The lymph channels all through the nodes were packed with red corpuscles and lymphocytes; occasionally large flattened cells appeared. Many of the larger cells contained pigment. It was rare to see two or three nuclei. Some of the lymph channels were crowded with pigmented cells; the larger of these rarely contained red corpuscles or lymphocytes. Numerous irregular masses, stained blue by Gram's method, about four times as large as the lymphocytes, were found, which appeared to be some hyaline substance. Many groups of lymphocytes showing fragmented nuclei were noted. Occasionally a mass of cocci was seen, some of which when stained by Gram's method proved to be streptococci. The groups of cocci failed to show any relation to the cells having fragmented nuclei. Masses of cocci were also found inside of the veins, others in the capillaries. Certain veins showed thrombi composed of mixed layers of lymphocytes, red corpuscles, blood-plates, and fibrin. Sections of the abdominal lymph nodes revealed substantially the same changes as those made in the bronchial nodes (or glands), and sometimes large multinucleated phagocyte cells abounded. In certain cases the adipose and areolar tissue about the nodes contained numerous lymphocytes. Evidences of hemorrhages in and around these nodes were frequently discovered.

Spleen. In sections of the spleen it was found that the changes were similar to those in the lymph nodes. The capsule was normal. The Malpighian bodies were numerous, distinct, and enlarged, often being twice the normal size. They also contained normal-looking lymphocytes and great numbers of large flat cells. The splenic pulp was crowded with lymphocytes. There were occasionally fragmented nuclei as well as numerous cells with very large nuclei, or with several nuclei (two to five). These sometimes contained pigment. Mitosis, polymorphonuclear plasma cells, or mast cells were rarely seen in this or any other section. One very large phagocytic cell contained eight other cells with degenerated nuclei. Numerous masses of cocci appeared in the spleen pulp, and at times short chains could be distinguished. There were no eosinophilic cells.

Bone-marrow from the Sternum. On microscopical examination the marrow cells and nucleated red corpuscles were found comparatively few. Lymphocytes were present, but not conspicuous. Preponderance of small mononuclear cells, slightly larger than red corpuscles, could be seen. Some large cells resembling endothelium cells were discovered. The eosinophilic cells were few. There were some doubtful giant cells. Fragmentation of nuclei was not pronounced. Some groups of cocci were seen.

Heart. Many lymphocytes were seen. Fragmentation of nuclei was detected. The heart muscle was not remarkable. Single lymphocytes lay between the muscle cells. In the lymphoid spaces collections of minute lymphoid cells were discovered. No bacteria.

Lungs. No evidence of bronchitis, pneumonia, or pleurisy. The outer layers of the bronchi, especially the smaller bronchioles, as well as the alveoli, were richly infiltrated with lymphoid cells. There were hemorrhages in the submucosa of the main bronchus, and in the wall below the epithelium a cell infiltration, mostly consisting of lymphocytes, together with plasma cells. There was a similar increase of cells in the walls of the bronchial tubes. Lymphoid cell deposits were found between the mucosa and submucosa and throughout both layers. There were plasma cells mixed with red corpuscles in the mucous membrane, although less numerous than the lymphoid cells. Several thrombi of leucocytes and fibrin were discovered in the various arteries.

Liver. The capillaries were distended with (chiefly) red corpuscles. There was also a lymphoid infiltration extending along the portal spaces and pushing out between the liver cells. Inside of the capillaries emboli of micrococci were often to be seen, but there was no evidence of inflammatory action in their vicinity. Considerable light brown granular pigment was present in the liver cells.

Duodenum. The solitary follicles were composed of cells of the lymphoid type and plasma cells in moderate numbers, the last not striking. Between the glands the lymphatic tissue was but slightly increased.

Colon. The solitary follicles were large and numerous. In many of the lymph channels of the submucosa lymphocytes were found in large numbers. Evidences of hemorrhages and numerous bacteria, probably saprophytes, were found in the mucous membrane.

Kidneys. The lymphatic collections were most marked in the cortex at its junction with the pyramid. They were sometimes seen about the veins and, in rare instances, apparently in the sheath of the vessel. This was a marked feature shown in all sections examined. The convoluted tubes contained reticulated coagulum, which was also seen around the glomeruli. These were occasionally compressed. In many sections lymphocytes were seen between the cells of the tubules. The nuclei of the epithelial cell stained well.

Pancreas. The fibrous tissue surrounding and adjacent to the pancreas was œdematous; many loose red corpuscles were seen in it. There was some infiltration with small nucleated cells. No evidence of pigmentation from previous hemorrhages. The ducts and the glandular parts of the pancreas were normal. The cells of the infiltration were found to be large lymphocytes. Fragmented nuclei were only exceptionally seen. There was infiltration with lymphocytes close to the pancreas, resembling lymphoid tissue and apparently not connected with lymph nodes. No bacteria were detected.

Adrenal Bodies. No special change was noted and no bacteria were found.

Thymus Gland. The connective tissue about the gland showed marked hemorrhagic infiltration, which appeared recent, as well as a few yellow pigment granules. Hassall's corpuscles were large and distinct. The thymus gland possessed numerous ordinary lymphoid cells. The distinction between cortex and medulla was barely discernible. The adjacent connective tissue contained lymphocytes, which seemed to pass

from the thymus proper to the adjacent tissue. A few eosinophiles were discovered. No fragmented nuclei were apparent; a few hyaline bodies were seen. The capillaries contained masses of streptococci.

A point worthy of note was that throughout the lymphoid structures appeared areas of nuclear degeneration and fragmentation. Although the nodes contained emboli of micrococci, the areas of fragmentation showed no relation to the bunches of cocci. It may be presumed, therefore, that the degeneration was due to toxins formed by the micro-organisms rather than by direct action of the organism upon the cells.

This case is a striking illustration of leukæmic changes, especially in their relation to the skin. The tumor formations originated in the hypoderm and terminated in nodules, and were readily discovered by palpation. Frequently they would spontaneously disappear, or were compelled to do so under the influence of arsenic. Later, however, when they began to involve the superficial layers of the skin and to become elevated they manifested a disposition to become permanent—more so than when deep-seated. The color of the skin overlying the lesions was practically normal, while over those in which the superficial layers of the skin had become implicated it was first red, then violaceous. These sequences, however, were not absolute, for, occasionally, the several shades were tinged with hemorrhagic streaks. The size of these deep-seated nodules was that of a hazel-nut. The deeply buried lesions were practically oval; the superficial ones were also oval, but were more or less flattened, or pad-like, and covered a considerable surface.

Cases of leukæmic infiltration of the skin have been recorded in medical literature since 1876, first by Bisiadecki,[1] followed by Hochsinger and Schiff,[2] Philippert,[3] and Oliver.[4] Most prominent among the more recent reports of excessive leukæmic growth may be mentioned the case of Kreibich,[7] who reports that the pendulous masses, which varied in size from a pigeon's egg to the adult fist, were found upon the face. The history of the majority of cases recorded shows that this locality is the common seat of leukæmic invasion. However, in the case under consideration, they predominated upon the chest. In referring to their frequent occurrence upon the face, Pinkus[6] argues that the explanation lies in the fact that this part of the body lacks protection from external injuries, or that it may be due to a better blood-supply. From my own observation of the present case I venture the opinion that it sustains the theory of infection. In this instance the infection possibly originated at the site of the initial appearance of the disease, which was upon the forehead, and it took place long before there was any lymphatic enlargement. From the excellent résumé of Pinkus it would appear that all such tumor-like formations occurring in the skin are clinically and histologically identical, for he makes no distinction between those found in lymphatic leukæmia and those in pseudoleukæmia. Microscopically, the cutaneous alterations of the

present case correspond with the findings of Kreibich, Nékám,[3] Pinkus, and Oertel,[5] who considered the microscopical structure analogous to lymphocytes deposited in the skin. In the case now discussed there was no marked suggestion of mitosis. The cells in size and form were identical with the lymphocytes, and were usually discovered within the walls of the vessels. It was observed that the cells were first lodged within the meshes of the corium, in close relationship to the subcutaneous tissue. The fact that a small number of lymphocytes were congregated about the bloodvessels in apparently normal skin—observed by Kreibich—was also noticed in the present instance.

One of the most striking features of the case, in the earlier stages of the disease, was the pronounced change in the complexion. The countenance assumed a vivid tinge of pink that challenged attention. This abnormal hue was undoubtedly of a hæmatogenous character. Another symptom of note was the dual pigmentation, depending upon several causes. First, we had the idiopathic pigmentation, which appeared early in circumscribed areas, while the physical condition of the patient was still unimpaired. Its initial color was yellow, but the older the areas the darker they grew. There was no inclination on their part to coalesce. Second, we had a secondary pigmentation which was more or less general, covering the entire chest and back to such an extent as to efface all traces of the first, existing in discrete and isolated patches. This was purpuric in character, and appeared in various shades of brown and black.

In reviewing the blood examination many interesting points may be recorded.* The most noteworthy were the blood changes, apparently demonstrating the development of lymphatic leukæmia from pseudoleukæmia, and showing that the blood upon the first examination was practically normal, not even revealing any marked diminution in the hæmoglobin. It was only when the case was advanced that a leukæmic condition was determined. However, the apparent transition of the pseudo type to that of the lymphatic was quite abrupt. The trans-

* While the title of this paper sufficiently indicates the writer's own interpretation, he is, nevertheless, fully conscious that there is room for a legitimate difference of opinion concerning the case. That could hardly be otherwise, so long as the classification of lymphosarcoma and pseudoleukæmia is so indefinite and unsatisfactory as at present. The question is sure to arise whether the facts of this case really justify the diagnosis of pseudoleukæmia developing into true leukæmia. The blood was at first normal, without leucocytosis or relative lymphocytosis It subsequently changed to the picture of leukæmia, with leucocytosis and high relative lymphocytosis—96 per cent. lymphocytes. The affection at first was either pseudoleukæmia or aleukæmia. Considering the limitations of our knowledge regarding the occurrence of an aleukæmia in the presence of physical signs of leukæmia (*i. e.*, glandular swelling) as a preleukæmic stage of true leukæmia, we prefer to take our stand on the simple facts of the case and say that the case was at first not true leukæmia, but pseudoleukæmia. Our knowledge of this condition may, in the future, be so enlarged that we shall be required, before reaching a positive diagnosis of pseudoleukæmia by blood examination, to hold the case under observation for a considerable length of time, in order to exclude the possibility of the existence of an aleukæmia preleukæmia.

mutation of pseudoleukæmia into genuine leukæmia is known, but is exceedingly rare. Reference may here be had to the cases reported by Mosler,[9] Fleischer and Pentzoldt,[10] Troje,[11] and Cabot.[12] Pentzoldt mentions a form of lymphatic Hodgkin's disease different from the ordinary variety of pseudoleukæmia which may result in true leukæmia. In comparing the first blood examination with the second the only noticeable change was in the number of leucocytes with secondary anæmia—the principal argument for the separation of pseudoleukæmia and lymphatic leukæmia. The same augmentation was manifest in the second and subsequent examinations. The only difference was in quality. After the second analysis the alterations in the skin showed the same clinical features. There was apparently the same general swelling of the lymph glands, the same cutaneous pigmentation and tumor formation, with the absence of any special enlargement of the spleen or any other internal organ. However, the patient appeared somewhat weaker, with a slight pallor of the visible mucous membrane, sufficient, however, to demonstrate that some important changes had taken place, and that we were dealing with a different type of leukæmia than that found at the first examination. There was a preponderance of leucocytes and an increase of the mononuclear elements over the polymorphonuclear neutrophiles, and a lack of myelocytes, all clearly certifying to the presence of lymphatic leukæmia. The further progress of the leukæmic stage showed a rapid loss of red corpuscles. The nucleated red cells also decreased, but, as the disease progressed, there was a considerable increase in the macrocytes. There was an enormous augmentation in lymphocytes, both relative and absolute, 95 per cent. of the 34,000 being small lymphocytes. A striking peculiarity of the case consisted in the fact that it ran its course with singular fidelity to its type until about one week before death, when severe soreness of the throat supervened, accompanied by grave constitutional symptoms. The temperature became unusually high, with rapid loss of strength, suggesting general infection. The enlargement of the lymphatic glands suddenly subsided, especially as concerned those about the neck, so that at the time of the patient's death they could only be detected by palpation. The blood showed, associated with this secondary infection, a drop from 45,000 cells to 600 per cm., with an increase in the proportion of polymorphonuclear neutrophile cells of 3.4 per cent. to 10 per cent., and a decrease in the proportion of lymphocytes of 95 per cent. to 88 per cent.—all within eight days. The infection was due to the streptococcus pyogenes, a fact corroborated by the bacteriological examination of secretions taken from the throat just before death, and verified by subsequent examinations made at the time of the post-mortem. The smeared preparations not only revealed the streptococcus, but it was also cultivated from the tissues procured from

the various organs of the body. It has been recognized for many years that the most striking changes in leukæmia occur following secondary infections, to such an extent that all features of the blood are altered. Herrick[14] reported a case of lymphatic leukæmia with a streptococcus infection beginning in the tonsils which was very much like the case under consideration. In acute leukæmia apparent improvement has been noticed, especially marked as to the leucocytosis, as the infection developed. In all probability it does not prolong life, as has been suggested, but only acts as a moderating agent for the time being. It is interesting to note the analogies which these phenomena present with the disappearance of sarcomata after the injection of the toxins of the streptococcus, as reported by Coley. Of the two cases reported by Fraenkel,[15] one on March 6th showed the presence of 89,000 leucocytes, consisting principally of lymphocytes. On March 21st the streptococcus infection was discovered, and on March 26th the number of leucocytes had decreased to 6000. In the second case following the infection the leucocytes decreased from 22,000 to 1200, yet death was the ultimate result in both cases. In other well-known recorded cases, as, for instance, Muller,[16] the leucocytosis fell from 246,900 to 57,300; in Kovács,[17] from 67,000 to 17,000; in Zeissl's,[18] from 140,000 to 9350; Cabot's, from 40,000 to under 500; Kormoczi, from 20,000 to 1000. In most of these cases there was also a diminution in the size of the spleen.

The unusual increase of the uric acid eliminated in the present instance, notwithstanding the fact that it corresponded with that of the cases previously observed, deserves special mention. The urine was carefully studied during the last few days of the patient's life, after the intercurrent infection had become active. Previous examinations had shown the constituents of the urine to be about normal. The greatest amount voided at any time during twenty-four hours occurred between noon of July 28th and that of July 29th, being 3240 grammes, containing 4.94 grammes of uric acid. This unusual increase took place when the sepsis had reached its height. There was no special attention given to the quantity or quality of food throughout the whole course of the disease, other than for the last few days. After the onset of the sore-throat the patient took, daily, about three pints of milk, six ounces of whiskey, and four eggs, with an unlimited amount of water to quench the thirst accompanying the high fever.

There seemed to be a marked relation between the elimination of uric acid and the loss of lymphocytes in the blood, although the sudden decrease of all lymphatic gland structures must also be taken into consideration as well as the food. In this connection attention is directed to the areas showing nuclear degeneration found in the bronchial and other lymph nodes. An increase in the elimination of uric acid is, as

a rule, noted in all cases of acute leukæmia ending in rapid destruction of the white cells, due to the sepsis or to infection. A. Magnus Levy[19] has recorded the most remarkable phenomenon of this character, viz., 8.72 grammes in twenty-four hours. He also observed in the same case, as well as in a number of other acute cases, that there was a large amount of urine and an increase of total nitrogen and uric acid.

I desire to express my sincere thanks to Dr. Herbert U. Williams for his kind assistance in the preparation of this article, and to Dr. Albert Woehnert for making the blood examinations.

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A CONTRIBUTION TO CLINICAL OBSERVATIONS ON BLOOD-PRESSURE.

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The vagueness which has long surrounded terms used to denote variation in blood-pressure has led men of an inventive turn of mind to devise instruments for determining these variations with some degree of accuracy. Special interest has been aroused within the past few years by a decided advance in such instruments which are of clinical value especially by the sphygmometer devised by Messrs. Hill and Barnard, of St. Bartholomew's Hospital, London, and the tonometer of Gaertner. The results of the experiments detailed in this paper have been determined by means of the sphygmometer; and although not an ideally perfect instrument, the results obtained with it are sufficiently uniform and accurate to be of distinct value in following cases from day to day, in keeping records of blood-pressure, and in making valuable observa-

tions. The manometer tubes will not soon be replaced in the experimental laboratory; but, of course, their usefulness is *nil* in gaining knowledge of altered blood-pressure at the bedside, to whatever causes due. The sphygmometer is in reality the manometer tubes reduced to a dial (on the principle of the aneroid barometer) and made practical for clinical use.

For those unfamiliar with the instrument a short description of the main points in its construction and method of use will be of interest.[1]

"It consists of (1) a broad armlet, which is strapped about the upper arm. The armlet is formed of a leather or flexible steel band, on the side of which there is fastened a bag of thin India-rubber. The rubber bag is connected by a Y-tube with (2) a small compressing air-pump fitted with a valve and (3) a pressure gauge. The pressure gauge is of special construction. Roughly, it consists of a metal tambour, the expansion of which is exhibited in a highly magnified form by means of an index or pointer which travels around a dial. This dial is graduated in millimetres of mercury."

Method of Use. The armlet, with the rubber bag, is fastened about the arm just above the elbow (or on any part of the arm or leg where it can be made to fit snugly). Air is now forced into the bag by means of the air-pump until the needle begins to move on the dial (there then being a continuous column of air between the bag and the registering mechanism in the pressure gauge). The pulse wave is communicated from the artery to the needle through the column of air already referred to, and is shown by an oscillating movement. The width of the needle's excursion increases with the increasing pressure in the bag until this pressure is equal to the mean arterial pressure, when the excursion of the needle will be at its greatest. The reading in millimetres of Hg on the dial will represent the mean arterial tension. From this point on, with increase of pressure in the bag, the excursion becomes less and less, and finally ceases at a point which corresponds to the maximum arterial pressure. It is, however, only the mean arterial pressure with which we have to do in these experiments.

In order to obtain accurate readings the patient must be at rest in the prone position, as it has been clearly demonstrated by Hill and Barnard:

"1. That the arterial pressure is the same in all the larger arteries while the subject lies horizontally.

"2. In changing from a horizontal to the vertical position the pressure in the upper part of the brachial artery (and it may be assumed also in the carotid artery) either remains constant or becomes slightly elevated.

"3. On changing from the horizontal to the sitting posture the pressure in the arteries of the lower limbs becomes higher than that in the upper limbs by the height of a hydrostatic column of blood separating the two points of observation. If the body remain horizontal and the legs are elevated the converse result holds true."[2]

These results have been proven true in these experiments, except the third point referred to, the correctness of which is assumed from Hill

[1] Hicks. Notes on the Sphygmometer.

[2] Proceedings of the Physiological Society, December 11, 1897.

and Barnard's observations. The difference, in the sitting posture, of the mean arterial pressure in brachial and peroneal arteries ranges from 10 to 25 mm. Hg, the average being 17 mm. Hg higher in the latter.

The armlet is best applied snugly and the reading repeated several times. In a certain number of cases the readings are difficult to obtain, as the oscillations of the needle are very nearly the same over quite a large area—*i. e.*, 5 to 10 mm.; in these the average of several readings must be taken.

Of course, one of the chief advantages of some such method of recording blood-pressure is that it does away with vague terms as "slight," "moderate," or "marked" increase in tension, which necessarily exist where the old method of relying entirely on the touch for one's information is used; for while an individual observer might be able to tell very accurately the relative difference in pressure from day to day, and his interpretation be correct so far as it went, his observations would be of little positive value or have little positive meaning to anyone who could not feel and interpret that particular artery for himself. Hence something definite is conveyed to the mind of anyone reading of a case of nephritis, for example, where the mean arterial pressure is recorded as 150 mm. Hg, knowing the average normal range to be 110 to 120 mm. Hg. Besides which it enables us, in chronic cases associated with increased blood-pressure, to find the drug which will do the maximum of work with the minimum dose—a point of considerable value when it is taken into consideration how long some of these cases require medication.

It is quite without the province of such a paper as this to discuss the various etiological factors in the production of increased arterial tension, and whether such variations are due to changes in the force of the heart's action, contraction of the arteries, thickened or sclerosed arteries, or, again, to direct changes in the composition of the blood itself (aside from the presence of toxic materials which stimulate the vasoconstrictors). For all such discussion one must refer to some of the standard works on the subject. Suffice it to say that changes in arterial pressure are due to any or all of these factors, and no doubt to others not so well recognized. This paper is strictly with reference to determining such changes as exist, from whatsoever cause, in an experimental way, and the modifications of pressure accomplished by certain drugs and procedures.

Of course, clinically, the classes of cases in which information in regard to increased blood-pressure is of the most value are those of nephritis, with or without arterio-sclerosis, and arterio-sclerosis without nephritis; but those cases with lowered arterial pressure are not to be lost sight of, and form an important class by themselves, to be dealt with later.

The number of cases of all descriptions whose blood-pressure was investigated was 129, and the total number of observations on pressure

503. As a routine in every case besides the pressure there was noted the urine examination, the relative amount of arterio-sclerosis present, and the medication in use.

For the normal healthy adult, Hill and Barnard place the average mean arterial pressure in young men at 110 to 120 mm. Hg in the standing position and 100 to 110 in the recumbent. The average pressure for the cases here recorded in the recumbent position was: Males 116 mm. Hg; females, 113 mm. Hg. The highest record of a mean arterial pressure noted was 235 mm. Hg; the lowest, 80 mm. Hg.[1]

For the sake of completeness the subject is divided as follows:

I. Nephritis. (*a*) Acute: without arterio-sclerosis; with arterio-sclerosis. (*b*) Chronic: without arterio-sclerosis; with arterio-sclerosis.

II. Arterio-sclerosis without nephritis: slight; moderate; marked.

III. Effects of some of the arterio-dilators—drugs and phlebotomy.

IV. Blood-pressure in various diseases.

V. Conclusions and résumé.

I. Nephritis.

(*a*) *Acute Nephritis.*

		All 9 cases.	*5 cases uncomplicated.*
With arterio-sclerosis 9 cases. Total number of observations and readings 53.	Highest mean arterial pressure	150 mm. Hg.	150 mm. Hg.
	Lowest " " "	82 " "	100 " "
	Average " " "	115 " "	123 " "

		All 7 cases.	*There were none uncomplicated.*
Without arterio-sclerosis 7 cases. Total number of observations and readings 50.	Highest mean arterial pressure	155 mm. Hg.	
	Lowest " " "	85 " "	
	Average " " "	117 " "	

A glance at this tabulated series of cases of acute nephritis shows one that, taking them as a whole, the highest, lowest, and average mean arterial pressure of cases of both classes—*i. e.*, with or without arterio-sclerosis—is almost identical. The five cases of nephritis with arterio-sclerosis uncomplicated by any other conditions, such as acute infectious diseases, etc., show an average pressure rather higher than that of all the cases taken together. This is accounted for by the fact that among the latter are included several cases with extremely lowered vitality, such as lobar pneumonias on the sixth to eighth day, marked anæmias, etc. In no case of a secondary parenchymatous nephritis complicating an acute infectious disease was the pressure over 122 mm. Hg, and it was usually much below this, ranging from 84 to 118 mm. Hg.

In all the cases in which arterio-sclerosis was present in conjunction with the nephritis it was slight or of only moderate degree, and in no

[1] For all discussion in regard to the variations in pressure produced by physiological processes, *e. g.*, sleep, exercise, emotion, etc., one is referred to Oliver's excellent article, Edinburgh Medical Journal, 1898, vol. iv. p. 1.

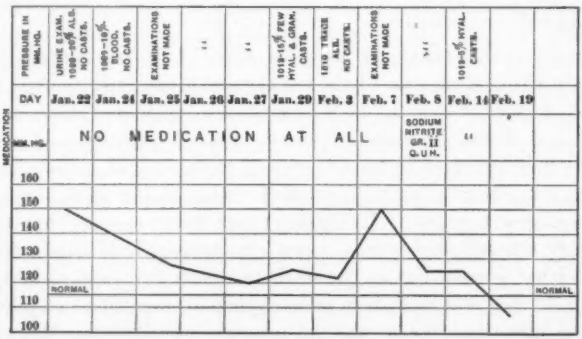
case were the arteries stiff and hard; then too, that the highest, lowest, and average pressures were practically the same in the two classes of acute nephritis cases—*i. e.*, with or without sclerosis—shows that slight or moderate arterial thickening plays comparatively a small rôle in the production of increased peripheral tension, a point which will be more clearly demonstrated when dealing with cases of arterio-sclerosis as a whole.

Another noteworthy fact in regard to these cases is that while we often get a great increase of blood-pressure under any conditions, we may find it normal, or, indeed, subnormal. In the five cases of acute nephritis without other complication than a slight or moderate arterio-sclerosis the average tension was 7 to 10 mm. Hg above the normal range.

It was found on examination of the urine in these cases that, on the whole, the greater the amount of albumin present the higher was the mean arterial pressure. This, however, had some notable exceptions, although they were almost invariably found among the cases with greatly lowered vitality from other causes.

Cases of Special Interest.

Case XXXVII.—Man, aged twenty-five years. No previous nephritic history. Patient has been a steady and heavy drinker for a number of years. Illness began about ten days previously, with loss of appetite, slight fever, and œdema of legs and face. Complained of very slight dyspnœa on exertion. Urine examination on admission: specific gravity, 1012; 20 per cent albumin. Blood: no casts; mean arterial pressure, 150 mm. Hg. The subsequent run of the case is shown by the accompanying chart:



This case shows the normal decline in blood-pressure which goes hand-in-hand with a spontaneous recovery from an acute diffuse nephritis, and, incidentally, the effect of one of the most reliable arterio-dilators. On one occasion there was an abrupt rise in pressure of about 30 mm. Hg without known cause. This fell promptly to normal as soon as sodium nitrite—2 grains every four hours—was given the patient.

There is one case of interest, showing, as it does, the return to normal from a lowered blood-pressure, as the result of exhaustion, on the ninth day, of a lobar pneumonia. The pressure then was 98 mm. Hg. Three days later—*i. e.*, after defervescence—pressure 105; and seven days later pressure 120 mm. Hg, while the patient's general condition had improved with tonics only as medication, the urine in the meantime becoming normal. The nephritis in this case only amounted to a slight transient albuminuria (1 per cent.), due to the acute parenchymatous degeneration complicating the pneumonia.

Further than these observations there was nothing of particular interest to note except the effects of medication on lowering pressure in these cases and a comparison of the averages with those of the cases of chronic nephritis with arterial sclerosis and those of arterial sclerosis without nephritis. This may be done at a glance, the points of comparative interest being brought out in the résumé.

(*b*) *Chronic Nephritis.*

		All 26 *cases.*	19 *cases uncomplicated.*
With arterio-sclerosis 26 cases. Total number of observations and readings 168.	Highest mean arterial pressure	235 mm. Hg.	235 mm. Hg.
	Lowest " " "	85 " "	112 " "
	Average " " "	177 " "	187 " "
Without arterio-sclerosis.	No cases seen.		

There were twenty-six of these cases of chronic nephritis observed, and all were complicated by an arterio-sclerosis of varying degree, there being none seen without it; and so far, as so limited a number of cases can, it shows that with chronic nephritis it is the rule to find this complication. However, as we shall see, the converse is not true, for many cases of arterio-sclerosis were observed without evidences of nephritis.

In looking over the cases as a whole the highest pressure found—235 mm. Hg—was in a case in which the urine showed a specific gravity of 1014, with 10 per cent. of albumin without casts, but complicated by one of the most extreme degrees of arterio-sclerosis found. No doubt here the sclerosis was much more the cause of the high pressure than the nephritis, the patient being comparatively comfortable in spite of it. This comparative comfort was usual in the arterio-sclerosis cases.

The lowest mean arterial pressure seen in this class was 85 mm. Hg, and was in a case of chronic diffuse nephritis in which the urine examination was quite unexpected. The specific gravity ranged from 1012 to 1031; the albumin 60 to 99 per cent., with a few hyaline and granular casts, and the patient passing 41 to 104 ounces of urine in the twenty-four hours. Here, however, the arterio-sclerosis was very slight; but it shows that a low arterial pressure may accompany a nephritis of considerable extent. In only one case of the series was the pressure found lower, and that was in the case already referred to with pericar-

ditis, rheumatism, and anæmia—pressure 82 mm. Hg. In the acute case as well the arterio-sclerosis was extremely slight.

A comparison of the pressures in these cases of chronic nephritis with those found in the acute condition shows the highest and average mean arterial pressure to be much higher in the former, the average mean arterial pressure being 62 mm. Hg higher in the chronic form. There is comparatively little difference in the lowest pressures to be found in each, either complicated or uncomplicated (arterio-sclerosis is ignored as a complication in this connection).

After careful examination of the data it is found that blood-pressure, urine examination, and the amount of arterio-sclerosis seem to bear no definite relation to each other beyond the fact that marked arterio-sclerosis complicating the nephritis increases the pressure.

Chronic Nephritis—Cases of Special Interest.

CASE L. was of interest as showing the lowering of blood-pressure during the period of apnœa in a patient with Cheyne-Stokes respiration in the terminal stages of chronic nephritis with chronic uræmia. Pressure after several deep inspirations, 140 mm. Hg; during apnœa, 125 mm. Hg. This difference was also appreciable to the finger, although the exact amount could not be accurately gauged in that way.

CASE LXXIX. illustrates the low pressure in cases of aortic regurgitation complicated by chronic nephritis. Patient, aged forty-five years; suffering from chronic nephritis. Urine: specific gravity, 1017; 20 per cent. albumin; granular and hyaline casts. Pressure, 85 mm. Hg. As will be shown under "Endocarditis," low pressure is the rule for aortic regurgitation, with or without nephritis or other complications, save where there is an accompanying mitral regurgitation.

CASE LXXXIV.—Acute pericarditis and articular rheumatism. Chronic nephritis; anæmia. Blood-pressure, 82 mm. Hg. Pericardium tapped; 3 ounces of bloody fluid obtained. After tapping, pressure 82 mm. Hg. No effect on blood-pressure whatever.

Under heading on "Effect of Drugs" other points of interest about these cases of chronic nephritis will be brought out.

II. ARTERIO-SCLEROSIS WITHOUT NEPHRITIS.

		All 18 *cases.*	7 *uncomplicated cases.*
Slight arterio-sclerosis 18 cases. 36 observations and readings.	Highest mean arterial pressure	128 mm. Hg.	122 mm. Hg.
	Lowest " " "	80 " "	104 " "
	Average " " "	107 " "	112 " "

		All 14 *cases.*	7 *uncomplicated cases.*
Moderate arterio-sclerosis 14 cases. 28 observations and readings.	Highest mean arterial pressure	130 mm. Hg.	125 mm. Hg.
	Lowest " " "	80 " "	108 " "
	Average " " "	107 " "	116 " "

		All 4 *cases.*	2 *uncomplicated cases.*
Marked arterio-sclerosis 4 cases. 43 observations and readings.	Highest mean arterial pressure	205 mm. Hg.	205 mm. Hg.
	Lowest " " "	130 " "	138 " "
	Average " " "	165 " "	174 " "

Total averages for the thirty-seven cases of arterio-sclerosis of all degrees without nephritis: highest, 205; lowest, 80; average, 131 mm. Hg.

The divisions into slight, moderate, and marked arterio-sclerosis are, of course, merely arbitrary and determined by touch, and as such cannot be supposed to be exact; but after feeling a great many vessel walls one naturally divides the relative amount of thickening into these three classes, and although the limits of each are not clear-cut, still for practical purposes they are accurate enough.

By uncomplicated cases are meant all cases of arterio-sclerosis pure and simple, without acute conditions or valvular endocardial lesions, both of which are found at times to modify the arterial pressure.

A ready comparison may be made of arterial pressure between these classes of cases taken as a whole, or the uncomplicated alone, on referring to the foregoing classification. Referring only to the uncomplicated cases in each class, it is easily seen that the highest mean arterial pressure of cases with slight or moderate arterio-sclerosis is practically the same, the variation being only 3 mm. Hg in favor of the latter; the same holding true for the lowest and average pressures of the two classes, the average pressure in both being practically that of the normal individual. On the other hand, when we look at the pressures of the cases with marked arterio-sclerosis we see a great increase in all three, the average pressure being 58 to 62 mm. Hg higher than in the cases with a slight or moderate sclerosis. This is such a definite fact that it may with safety be laid down as a rule, that it is only an arterio-sclerosis of marked degree that, unaided by other conditions, increases to any extent the blood-pressure in the peripheral circulation. The same increase is shown in the cases taken as a whole, complicated and uncomplicated alike.

This failure of slight or moderate sclerosis to modify the arterial pressure was also shown in the cases of acute nephritis, to which the reader is referred.

The general idea gained in looking over standard works which treat of circulatory conditions in disease is that arterial thickening in general causes an increase in the blood-pressure of the individual—*i. e.*, no distinction is made as to the amount of the sclerosis necessary to cause such a result. This may be true theoretically, and to some extent practically, so far as the internal blood-pressure is concerned, but as yet we have no means of estimating such a condition if it really exists; certainly the peripheral blood-pressure is not materially increased except in cases in which the sclerosis is extreme. The number of cases of slight or moderate arterio-sclerosis in this series seems large enough to formulate the foregoing rule with safety. Those cases which were of especial interest in these classes will be discussed under the head of

their associated complications. None of the cases of straightforward, uncomplicated arterio-sclerosis were of particular interest.

III. Effect of some Drugs and Procedures Used in the Reduction of Increased Arterial Pressure.

Clinically, the effects of medication are often perfectly plain, and we do not need the evidence of any new method, such as sphygmometry, to convince ourselves that certain arterio-dilators do good under certain conditions associated with increased peripheral blood-pressure. What is of interest, however, in this connection is:

1. How much effect do these drugs really have, measured by some definite means?
2. How promptly do they act?
3. How long does their action continue?

As this series of experiments is only meant to be a contribution to the study of the effects of some of the arterio-dilators, exhaustiveness is not claimed, but further knowledge is to be gained by future investigation.

It has been a difficult matter to determine satisfactorily, in many instances, what particular drug had the most marked effect in the reduction of blood-pressure, even with the aid of an instrument, and the results of observation here given have been determined with much difficulty. In order to obtain results as uniform as possible, with few exceptions the observations were made at the same time of day, viz., between 2 and 5 P.M., so that taking of food, exercise, etc., should not play a rôle in the variations, the patients being at rest in bed.

As will be seen by reference to the tables, the promptness of the drug's action and its duration was noted, while in others the general continued effect was observed. It has also been attempted to show the direct effect of medication and the pressure before administration of the drug; afterward the length of time before the maximum effect was produced, the duration of effect of a single dose, and any clinical points of interest about the cases, drawing conclusions, so far as possible, as to efficacy.

Erythrol Tetranitrite. In two cases the drug was given by the mouth, ½ grain t. i. d.

First case, marked arterio-sclerosis. Pressure before administration, 150 mm. Hg; after twenty-four hours' use, pressure 140 mm. Hg.

Second case, chronic nephritis, with marked arterio-sclerosis. Pressure before, 145 mm. Hg; after twenty-four hours' use, pressure 135 mm. Hg.

Erythrol tetranitrite reduced pressure in each case 10 mm. Hg. The drug is too expensive for general use and not as effectual as sodium nitrite.

NITROGLYCERIN. SERIES OF TWELVE CASES.

No.	Diagnosis.	Dose of nitroglycerin.	How given.	Pressure before.	Pressure after.	Effect noted in	Amount.	Effect lasted	Pressure returned to	Urine examination.	Amount of arterio-sclerosis.
1	Chronic nephritis, acute exacerbation,	Gr. $^1/_{600}$ every 2 hrs.	By mouth	155 mm. Hg.	125–130 mm. Hg.	Within 24 hours.	25–30 mm. Hg.	Continued to fall during convalescence.	.....	1023, 25 per ct. casts; 1020, 2 per ct. in two days.	Slight.
2	Acute nephritis,	Gr. $^1/_{100}$ every 2 hrs.	Hypo.	155 " "	130 mm. Hg	Within 24 hours.	25 mm. Hg.	Four days, while on drug.	Continued to fall.	1025, 60 per ct. blood casts; 1010 trace casts.	None.
3	Abdominal tumor,	Gr. $^1/_{100}$ once.	Hypo.	122 " "	115 " "	9 min.	7 " "	1½ hours.	Not seen.	1021, o. k.	None.
4	Acute nephritis,	Gr. $^1/_{60}$ once.	By mouth.	118 " "	105 " "	20 "	13 " "	1 hr. 20 min.	115 mm. Hg.	1018, 2 per ct. casts and blood.	Slight.
5	Cirrhosis of liver,	Gr. $^1/_{60}$ once.	By mouth.	115 " "	110 " "	15 "	5 " "	About 1 hour.	115 " "	1015, hyaline casts.	Slight.
6	Chronic nephritis, acute exacerbation,	Gr. $^1/_{60}$ once.	Hypo.	150 " "	135 " "	15 "	15 " "	1 hour.	150 " "	1020, 80 per ct. casts and blood.	Slight.
7	Lobar pneumonia, chronic nephritis,	Gr. $^1/_{100}$ once.	By mouth.	150 " "	150 " "	None.	0	0	.....	1017, 30 per ct. casts.	Moderate.
8	Marked arterio-sclerosis,	Gr. $^1/_{100}$ once. Gr. $^1/_{100}$ every 1 hr.	Hypo Mouth.	200 " " 200 " "	200 " " 200 " "	None. None.	0 0	0 0	.....	1026, negative.	Marked.
9	Chronic nephritis,	Gr. $^1/_{100}$ every 2 hrs.	Hypo.	122 " "	122 " "	None.	0	0	.....	1020, 5 per ct. casts.	Moderate.
10	Gastroduodenitis, jaundice,	Gr. $^1/_{60}$ once.	By mouth.	105 " "	105 " "	None.	0	0	.....	1020, negative.	None.
11	Lobar pneumonia,	Gr. $^1/_{100}$ every 4 hrs.	By mouth.	108 " "	111 " "	None.	0	0	.....	1030, negative.	Sligh .
12	Chronic nephritis,	Gr. $^1/_{100}$ every 2 hrs.	By mouth.	112 " "	112 " "	None.	0	0	.....	1016, 30 per ct. casts.	Moderate.

Conclusions. 1. Continued effect of $\frac{1}{100}$ grain, by the mouth or hypodermically, results in lowered pressure, sometimes very good, and reduction may be greater than after a single dose under similar conditions. This is a fact in clinical experience where tension is high—*e. g.*, Cases I and II.; uncertain where tension is moderate—*e. g.*, Case IX.

2. The direct effect of a single dose of $\frac{1}{100}$ grain hypodermically is uncertain, sometimes resulting in moderate reduction of pressure—*e. g.*, Case III.—or is ineffectual. Case VII. (here the patient died in uræmic convulsions, phlebotomy even failing to reduce pressure).

3. The direct effect of a single dose of $\frac{1}{50}$ grain is reliable, with moderate lowering of pressure—5 to 15 mm. Hg.—in fifteen to twenty minutes. The promptness of action is about the same whether given by the mouth or hypodermically—*e. g.*, Cases IV., V., and VI.

4. The average duration of the positive effect from any dose is one hour and twelve minutes. No lowering of pressure when increase is due to marked arterio-sclerosis, either by a single dose of $\frac{1}{100}$ grain hypodermically or continued every hour by the mouth—*e. g.*, Case VIII. When pressure is already subnormal no further lowering follows the use of a single dose of grain $\frac{1}{100}$, or every two hours or every four hours—*e. g.*, Cases X., XI., and XII.

Résumé of Cases Given Sodium Nitrite. Dosage, ½ to 3 grains, always by the mouth.

Case XIII.—Most marked reduction of pressure in case of extreme arterio-sclerosis—50 mm. Hg. (This was repeated with identical results.)

Taking the other cases with positive results we find: Pressure lowered 10 to 15 mm. Hg; average, 12.2 mm. Hg. Effect evident in ten to fifty minutes; average, twenty-six minutes. Effect lasted one and a half to two hours; average, one hour and forty minutes.

The cases were acute or chronic nephritis, with more or less arterio-sclerosis, and one case of marked arterio-sclerosis without nephritis. One negative result. The case was a chronic nephritis, with an acute exacerbation and uræmic convulsions, and required phlebotomy, sodium nitrite having no effect.

Conclusions. Sodium nitrite reduces arterial pressure promptly, and is usually best given in doses of 2 to 3 grains every two to four hours, depending on the urgency of the symptoms. According to these experiments this is the most satisfactory and reliable arterio-dilator, and acts whether the increased pressure is due either to nephritis or arterio-sclerosis. As with nitroglycerin, continued use in some cases shows greater effect than a single dose. No depressing effects on the patient's general condition were seen.

Potassium Iodide. Four cases tested. Dosage, 10 grains t. i. d. in three cases, and 5 grains every four hours in one case. No effect from either could be discovered by the sphygmometer.

SODIUM NITRITE. SERIES OF ELEVEN CASES.

No.	Diagnosis.	Dose of sodium nitrite.	How given.	Pressure before.	Pressure after.	Effect noted in	Amount of reduction.	Effect lasted	Pressure returned to	Urine examination.	Amount of arteriosclerosis.
13	Marked arteriosclerosis,	Gr. ij every 4 hrs.	By mouth.	200 mm. Hg.	150 mm. Hg.	Within 4 hours.	50 mm. Hg.	While taking drug.	200 mm. Hg. 8 hrs. after last dose.	1026, negative.	Marked.
14	Normal case,	Gr. j every 4 hrs.	" "	110 " "	95 " "	8 hrs after first dose; not seen before.	15 " "	Not noted.	.....	1032, negative.	Moderate.
15	Acute nephritis,	Gr. ij once.	" "	125 " "	112 " "	30 minutes	13 " "	2 hours.	125 mm. Hg.	1011 trace alb. gran. casts.	Moderate.
16	Acute nephritis,	Gr. ij once.	" "	128 " "	112 " "	50 "	13 " "	2 hours.	128 " "	1011, trace, casts.	Moderate.
17	Acute nephritis,	Gr. ij once.	" "	128 " "	112 " "	10 "	13 " "	1 hr. 50 min.	128 " "	1027, 3 per ct. albumin, hyal. casts.	Slight.
18	Chronic nephritis,	Gr. ½ once.	" "	122 " "	110 " "	15 "	12 " "	1 hr. 30 min.	Not noted.	1013, 3 per ct. albumin, no casts.	Moderate.
19	Chronic nephritis,	Gr. iij once, & every 4 hs.	" "	155 " "	140 " "	26 "	15 " "	1 hr. 30 min.	155 mm. Hg.	1017, trace, no casts.	Marked.
20	Chronic nephritis, acute exacerbation,	Gr. ij every 4 hrs.	" "	155 " "	155 " "	0	.....	.....	.....	1023, 75 per ct. albumin, casts.	Slight.
21	Chronic nephritis,	Gr. iij once, & every 4 hs.	" "	150 " "	140 " "	15 minutes	10 mm. Hg.	1 hr. 35 min.	150 mm. Hg.	1035, 5 per ct. casts.	Moderate.
22	Chronic nephritis,	Gr. ij every 4 hrs.	" "	150 " "	140 " "	Not seen until next day.	10 " "	.....	.....	1015, 35 per ct. casts.	Moderate.
23	Chronic nephritis,	Gr. ij every 4 hrs.	" "	122 " "	110 " "	Not seen until second day.	12 " "	While taking drug.	.....	1020, 5 per ct. albumin casts.	Moderate.

Chloral Hydrate. Three cases tested. Dose, 5 grains every four hours in all the cases. Diagnosis: Marked arterio-sclerosis; acute nephritis; chronic nephritis. No effect could be discovered. Tests were not conclusive.

Spiritus Etheris Nitrosi. Two cases. Dosage, ½ drachm, every four hours. No effect discovered.

PHLEBOTOMY AND INTRAVENOUS INFUSION OF HOT NORMAL SALT SOLUTION. TABLE OF FIVE CASES.

No.	Diagnosis.	Am't of blood withdrawn.	Am't of saline infusion.	Pressure before the operation	Pressure after the operation	Amount of fall in the blood pressure.	Remarks and notes on cases.
29	Chronic nephritis.	Oz. v.	1500 c.c. at 114° F.	200 mm. Hg.	150 mm. Hg. next day.	50 mm. Hg.	Marked general improvement; urine 1019, 30 per cent. albumin; casts.
66	Chronic nephritis.	Oz. viij.	1400 c.c.	195 mm. Hg.	150 mm. Hg. 3 days later.	45 mm. Hg.	Marked general improvement; urine 1013, 5 per cent. albumin; casts.
97	Chronic nephritis, marked arterio-sclerosis.	Oz. viij.	1200 c.c.	180 mm.	170 mm. next day.	10 mm. Hg.	The extreme arterio-sclerosis probably had much to do in preventing more complete fall of pressure; clinically patient much better next day, although tension still high.
40	Chronic nephritis.	Oz. vj.	1500 c.c.	122 mm.	122 mm. 6 hours after.	0	General condition much improved, although tension not lowered; urine 1020, 5 per cent. hyal. and gran. casts.
31	Chronic nephritis, convulsions.	Oz. xv.	1500 c.c.	125 mm.	125 mm.	0	Condition improved, though pressure not lowered; urine 1016, 20 per cent. alb.; red-cells.

Conclusions. Phlebotomy and saline infusion are good in all cases with markedly increased tension and where patient's condition is serious as a result of uræmic poisoning. In some cases blood-pressure is decidedly lowered, and in all there is marked improvement in the patient's general condition, even where no lowering of blood-pressure can be detected.

Effect of Extract of Suprarenal in Increasing Blood-pressure. Six cases. Dose, 5 grains, t. i. d. and every four hours. Results were the same in either dosage. Positive results in five cases. Pressure increased 2 to 10 mm. Hg; average, 6.6. mm. Hg. The effect was usually evident after two doses had been taken, and lasted while the drug was exhibited. One negative result. Case received 5 grains t. i. d. and every four hours. No effect was discovered, although the drug was continued for five days. In all these cases the blood-pressure was normal or subnormal before medication.

The positive effects of extract of suprarenal in increasing the blood-

pressure are sufficiently marked to open the way for a wider field of usefulness for the drug in conditions representing a lowered vascular tone. Its local effects are so well known that nothing need be said for its local vasoconstrictor stimulative power, but much wider experiment is needed to decide of just how much use it can be in conditions where it is desirable to increase the general blood-pressure.

IV. Notes on Blood-pressure in Various Diseases Other than Nephritis or Arterio-sclerosis.

Endocarditis—Valvular Lesions. One learns very little in regard to the working capacity of the heart by sphygmometry; and in no way can the sphygmometer compare with the sphygmograph for this purpose, for it is remarkable how serious a valvular lesion may exist and how desperately ill a patient may be and still have practically a normal blood-pressure. A striking example of this was a patient with double aortic lesion and mitral insufficiency, who was clinically in great distress, sitting upright in bed, breathing with difficulty, the heart markedly dilated and hypertrophied, in whom the arterial pressure was 115 mm. Hg—*i. e.*, normal.

The two points worthy of mention under this heading are: 1. The low pressure seen in cases of aortic regurgitation uncomplicated by any mitral lesion. Three cases of this kind were observed with pressures of 80, 85, and 85 mm. Hg, respectively; the regurgitation was undoubted. 2. The compensation (so far as pressure is concerned), in some cases of aortic regurgitation, by a mitral insufficiency.

Cases with aortic regurgitation and mitral insufficiency: (1) One with mean arterial pressure of 115 mm. Hg. (2) One with mean arterial pressure of 110 mm. Hg. (3) One with mean arterial pressure of 155 mm. Hg (complicated by nephritis).

The last case shows that the blood-pressure may be even away above normal in these cases if complicated by nephritis.

Lobar Pneumonia. There were twelve cases observed, and notes on pressure of the blood taken on the second, third, fourth, fifth, sixth, seventh, eighth, ninth, and tenth days. Nothing definite was discovered in regard to a continued rise or fall of pressure during the progress of the disease. Cases which showed a low pressure after the crisis (and this was quite frequently true) also showed a steady rise to normal pressure during convalescence. Observations of prognostic value were not made.

Anæmia (Primary). It was an interesting fact noted in this class that the blood-pressure was almost invariably lowered unless complicated by a nephritis or marked arterio-sclerosis. This is quite contrary to such authority as Oliver, who states that in chlorosis and anæmia mean

arterial pressure is, as a rule, raised, but the rise is generally but moderate in degree.[1]

TABLE OF CASES OF ANÆMIA.

Case.	Hæmoglobin.	Complication (if any).	Mean arterial pressure.
1	60 per cent.	Gastric ulcer	82 mm. Hg.
2	62 "	Acute rheumatism	82 " "
3	40 "	Gastric ulcer	84 " "
4	65 "	Gastric ulcer	85 " "
5	48 "	None	85 " "
6	65 "	None	85 " "
7	55 "	Gastric ulcer	85 " "
8	60 "	None	95 " "
9	65 "	None	95 " "
10	40 "	Gastric ulcer	95 " "
11	60 "	Catarrhal jaundice	105 " "
12	45 "	Gastric ulcer and acute nephritis	108 " "
13	85 "	None	110 " "

The only exception to this almost invariable rule of low pressure associated with anæmia is in Case XIII., and here the hæmoglobin was 85 per cent., or at least 20 per cent. higher than in any of the other cases; this percentage of hæmoglobin is so nearly that which is ordinarily found in most individuals as normal that it seems fair to class it as such. The accompanying nephritis in Case XII. accounts for the pressure—108 mm. Hg—with low hæmoglobin. No accurate relation between the amount of hæmoglobin and the mean arterial pressure could be discovered.

Gastric Ulcer. There were five cases of this condition observed, all complicated by a greater or less degree of anæmia. The blood-pressure ranged from 82 mm. Hg to 100 mm. Hg, the average being 87 mm. Hg. The hæmoglobin, estimated by Von Fleischl method, ranged from 55 per cent. to 70 per cent., the average being 67 per cent.

The thought which naturally arises in this connection is: Could not the lowered pressure, which we find almost always accompanies an anæmia, be a factor in the production of gastric ulceration? for, as is well known, nearly all the cases of ulceration show signs of anæmia before the symptoms of ulceration develop; at least, it seems that this may in some cases be proved to play a rôle in the production of gastric ulcer—*e. g.*, lowered pressure in the capillaries of the mucosa, partial or complete stasis in some, and diminished vitality of the part, with autodigestion at this point, resulting in ulceration.

No mention of such etiological factor can be found in any of the numerous general and special treatises on the subject consulted, but it offers at least a field for investigation.

Typhoid. Three cases. All showed lowered pressure when accompanied by high temperature.

[1] G. Oliver. Blood and Blood-pressure, 1901, p. 261.

Other Cases Noted. Trichinosis; cirrhosis of liver; acromegaly; Raynaud's disease; epilepsy; bronchitis, with or without asthma; rheumatism, acute or chronic; rheumatic purpura; tertian malaria; tubercular peritonitis; sciatica. In none of these cases was there anything abnormal found in regard to blood-pressure.

V. Résumé and Conclusions.

1. Average normal mean arterial pressure is: males, 116 mm. Hg; females, 113 mm. Hg.

2. In acute nephritis the accompanying arterio-sclerosis, if present, is of slight or moderate degree.

3. In acute nephritis the height of the blood-pressure varies, for the most part, directly with the amount of albumin in the urine.

4. An acute parenchymatous nephritis complicating an acute infectious disease causes little or no increase in blood-pressure.

5. The average mean arterial pressure in chronic nephritis is about 62 mm. Hg higher than that of acute nephritis.

6. There were no cases of chronic nephritis seen without an accompanying arterio-sclerosis.

7. In arterio-sclerosis it is only those cases in which it is extreme that the average mean arterial pressure is raised.

8. The most satisfactory drug for lowering blood-pressure, whether increase is due to nephritis or marked arterio-sclerosis, is sodium nitrite, best given in 2 to 3 grains every two to four hours. It acts in about twenty-six minutes, causing a reduction of 10 to 15 mm. Hg (in marked arterio-sclerosis sometimes much more), the effect lasting one hour and forty-four minutes on the average.

9. When the blood-pressure is high and is accompanied by symptoms of uræmia the best method of rapid reduction is phlebotomy—5 to 8 ounces, followed by saline infusion (1400 to 1500 c.c. of hot normal salt solution) and sodium nitrite in full doses. Clinically, also, there is almost invariably improvement.

10. Blood-pressure in aortic regurgitation, whether complicated by nephritis or not, is low. If a mitral regurgitation is also present pressure may be nearly normal.

11. Blood-pressure in all forms of anæmia is below normal.

12. Possible relation between lowered pressure in chlorosis and the production of gastric ulcer.

It is with a sense of the author's heartiest appreciation that he acknowledges the kindness of the medical attending and house staff of the Presbyterian Hospital, New York, for their co-operation in carrying out these experiments in the wards of that hospital; with especial thanks to Prof. Walter B. James and Dr. John S. Thacher, for their aid and encouragement in completing the work.

REVIEWS.

THE DISEASES OF THE RESPIRATORY ORGANS, ACUTE AND CHRONIC. By WILLIAM F. WAUGH, A.M., M.D., Professor of Practice and Clinical Medicine, Illinois Medical College, etc. Pp. 221. Chicago: G. P. Engelhard & Co., 1901.

A SMALL book, and one containing in its preface indications that the author possesses a virile belief in the efficacy of remedial measures, attracts attention. A belief that the function of the physician is neither one of expectancy nor yet of passive acquiescence in the natural progress of disease is hopeful, particularly when the method of intervention is not only clearly stated, but with its statement is given the reason for its recommendation. Without criticism of occasional printer's errors, the absence of index, infrequent vernacular in place of scientific diction, and even passing by some inconsistencies in text, we would call attention to the evidence, which the work contains, of positively good results to be obtained by the author's method as here exemplified. There is evidence that the book is written from the stand-point of the physician, realizing his responsibilities as well as his limitations, and not as indicative of a burning desire for literary fame. Of practical therapeutic indications the volume contains a host; of important, even if secondary, matters for attention, it is replete with suggestion. If we might join issue with the writer, we would suggest that with some potent remedies, of rather slow elimination, the dose-interval might be very properly prolonged in the interest of the patient. While we are given to understand that this is not to be regarded as "a finished product," when a more substantial work is presented we are inclined to believe that much more than "a stick may be retained in the final structure." We are to regard this only as a foundation upon which the author may erect a more thorough, a more complete, and, quite likely, a more æsthetic superstructure.

R. W. W.

A TREATISE ON ORTHOPEDIC SURGERY. By ROYAL WHITMAN, M.D., Chief of the Orthopedic Department of the Vanderbilt Clinic in the College of Physicians and Surgeons of Columbia University, etc. Illustrated by 447 photographs. Philadelphia and New York: Lea Brothers & Co., 1901.

THE author wastes no time in definition of orthopedic surgery or with general introduction to the subject, but plunges at once into a discussion of the conditions generally treated of under this head, and the first chapter is devoted to Tubercular Disease of the Spine. This general subject is treated by regions, the spine being divided for the

purpose into the lower, middle, and upper regions, with suitable treatment for lesions in each section. The complications of spinal tuberculosis are admirably dealt with, the author believing, for instance, that if abscess is not present when treatment begins one may hope to ward it off by suitable measures. Dealing with the subject of abscess, he evidently recognizes the difficulty of treating such cases radically, and yet is nevertheless more of a friend of operative intervention than are many of the so-called orthopedists, in which he shows a much riper surgical judgment. He advises against operation in the paraplegia of Pott's disease, his experience having shown its dangers as well as the safety, even if the tediousness, of non-operative treatment. He speaks kindly of Calot's forcible correction method for the deformity of Pott's disease, and describes it in detail. His plan of gradual correction is also given at considerable length and in most admirable manner. In the second chapter Non-tuberculous Affections of the Spine are fully considered, including syphilis, actinomycosis, acute osteomyelitis, and rickets. One of the most valuable portions of the book is that devoted to painful affections of the spine not attended by any deformity, such as those which result from typhoid fever and gonorrhœa, including, also, arthritis, spondylitis deformans, and particularly those painful affections which are not even attended by rigidity, such as the neuroses.

The subject of lateral curvature of the spine is well dealt with, these curvatures being classified in eight different varieties, according to their etiology, which shows how closely the author has studied the clinical and etiological aspects of these cases. He not only does ample justice to but cordially commends Teschner's system of gymnastic treatment with dumb-bells, while not failing to describe other perhaps less violent methods. Chapter IV. deals with other deformities of the chest and spine, including Sprengel's deformity of the scapula, asymmetrical development, etc. Wolff's classical researches into the pathogenesis of deformity have also full justice done them. Chapter V. concerns Tubercular Disease of Bones and Joints. Enough is said of their pathology to show that the author is familiar with it, although much is left to the special text-books. In Chapter VI. non-tubercular diseases of the same structures are dealt with, including syphilitic and post-gonorrhœal, with other forms of infectious arthritis, osteoarthritis, scurvy, and Charcot's disease. Chapter VII. deals with Hip Disease. In considering its mechanical treatment he discusses fairly the relative merits of traction with and without rest—*i. e.*, in bed or in splints and braces. The next chapter deals with non-tubercular diseases of the same joint, while the following three chapters concern the other major articulations. Chapter XIV. deals with Congenital Dislocation of the Hip, the author being decidedly inclined to view the lesion as being due to a defect in development. He seems to favor operation in all cases where radical cure is desired, and describes the three operative methods, namely, the open (Lorenz), forcible replacement, and intermediate operation, claiming that the prospect of success is directly related to the age of the patient. Dr. Whitman would advise that in children under six there should be first tried the functional weighting method of Lorenz, which, if ineffectual, may be followed by the open operation. Coxa vara is naturally dealt with in the same chapter, his view being that it is best treated by linear, or, if necessary, by cuneiform osteotomy.

Chapter XV. concerns Deformities of the Legs, such as genu valgum and varum and bowlegs, and the ensuing chapter deals with those of the upper extremities; then come two chapters concerning the important subject of diseases of the nervous system, which so often and so unfortunately lead to deformity, including poliomyelitis, the various paralyses, muscle dystrophies and atrophies, and hysterical and neurotic joints, the description and advice contained therein being of very great value. Chapter XIX. concerns the various forms of torticollis, the author giving exceedingly careful consideration to the indications for and the performance of the various operations called for in this condition. The concluding portion of the volume, some 140 pages, is given up to the various disabilities and deformities of the foot, including neuralgic affections and their especial treatment by improved footwear, and, in fact, comprising about everything that there is to be said in regard to these most annoying maladies.

One of the important features of the work as a whole is the not only frequent but judicious use made of statistics, the author seeming to have apportioned his space largely to the statistical importance of each subject dealt with. Such references as he makes to the surgical anatomy of parts involved are delightfully succinct and to the point, and of particular value is his enumeration of certain anatomical landmarks. Altogether, we know of no more admirable presentation of the salient practical points of orthopedic surgery than this volume affords, nor is there any work to which one may refer with more certainty of finding the latest and best. The author's large store of experience is evinced throughout the work, and his ripe judgment is especially that which makes the work so commendable. R. P.

THE TREATMENT OF FRACTURES. By W. L. ESTES, A.M., M.D., Director and Physician and Surgeon-in-chief of St. Luke's Hospital, South Bethlehem, Pa. Published by the International Journal of Surgery Co., 100 William Street, New York.

THIS book, which presents a large personal experience with fractures, possesses practical points of value not to be found in many of the more extensive works on this subject. The author shows and teaches a proper consideration of the patient's suffering, and constantly tells the attendant how this may be avoided or relieved—advice which is very much needed by the beginner. At the same time the most thorough methods of reduction and dressing are recommended. The pages devoted to "first aid, etc.," might be read with profit by everyone.

The author is an ardent advocate of plaster-of-Paris wherever it is possible to use it, and of its early application. We can hardly agree with him, however, as to the best time at which to begin massage and passive motion. This is a much-debated question, but the advocates of early motion and massage are certainly increasing in number every day. We feel confident that if early motion of muscles and joints were employed there would be a much earlier return of function than Estes is able to promise. This is particularly true of fractures near the wrist-joint.

The writer's attitude toward and experience with fractures of the spine in which the cord is injured is much in accord with that of the best authorities. A serious omission is that of the Jones dressing for fractures of the lower end of the humerus. The author recommends the early application in these cases of plaster-of-Paris from the fingers to the axilla, fixing the arm in a right-angle position. This dressing will not appeal to anyone who has employed the Jones position in the treatment of these fractures. We cannot commend the author's method of dressing a Colles fracture with plaster-of-Paris, although he does leave the fingers free and advises early motion of them This dressing he leaves undisturbed for two weeks, which we are inclined to think is bad treatment of the wrist-joint and its neighboring tendons.

As a piece of bookmaking we regret that we can say nothing commendatory, since the binding is poor and the type too small.

J. H. G.

PATHOLOGICAL TECHNIQUE. A PRACTICAL MANUAL FOR WORKERS IN PATHOLOGICAL HISTOLOGY AND BACTERIOLOGY, INCLUDING DIRECTIONS FOR THE PERFORMANCE OF AUTOPSIES AND FOR CLINICAL DIAGNOSIS BY LABORATORY METHODS. By FRANK BURR MALLORY, A.M., M.D., Assistant Professor of Pathology, Harvard University Medical School; First Assistant Visiting Pathologist to the Boston City Hospital; Pathologist to the Children's Hospital and to the Carney Hospital; and JAMES HOMER WRIGHT, A.M., M.D., Director of the Clinico-Pathological Laboratory of the Massachusetts General Hospital; Instructor in Pathology, Harvard University Medical School. Second edition, revised and enlarged. Pp. 432, with 137 illustrations. Philadelphia and London: W. B. Saunders & Co., 1901.

THE second edition of this justly prized manual shows considerable improvement over the first. The book is larger by 35 pages, the illustrations have been increased from 105 to 127, some sections have been rewritten, a number of new methods have been added, and some of the old methods have been revised. Among the important alterations are the amplification of the description of the parasite of actinomycosis, and the insertion of descriptions of the bacillus of bubonic plague, of the parasite of mycetoma (Madura foot), and of Wright's methods for cultivating anaërobic bacteria. Among the important additions are new staining methods for elastic tissue, by Weigert; for bone, by Schmorl, and for connective tissue, by Mallory. Most of the new illustrations are reproductions of photomicrographs of different bacteria. While it is distinctly stated in the preface that no attempt has been made to include in the book all methods and formulæ, but rather to exercise discrimination, and to select only those methods and formulæ that either have stood the test of practical experience or give promise of great usefulness, the book would have been made even better than it is had this rigidity been tempered a little by including more details of the Rokitansky method of performing necropsies, Jenner's blood stain, methods other than von Fleischl's for determining the percentage of hæmoglobin in the blood, details for detecting typhoid bacilli in the

urine, Lubarsch's gentian violet method for demonstrating glycogen in tissues (especially as Lubarsch's less reliable iodine-hæmatoxylin method has been incorporated in this edition), etc. These, however, are details. On the whole the book is most praiseworthy. It is a book that no one interested in pathological histology and bacteriology can well do without—a book than which there is no better on pathological technique. A. O. J. K.

INTERNATIONAL CLINICS. Edited by HENRY W. CATTELL, A.M., M.D., Philadelphia, U. S. A. Vol. II. Eleventh series. Pp. viii., 304. Philadelphia: J. B. Lippincott Co., 1901.

WE have on several occasions reviewed the different volumes of this series. The present contains important papers on locomotor ataxia, by Fournier, Frenkel, and Starr. A theoretical discussion entitled "Some Suggestions as to the Mechanism of Mental Operations," by Ramon y Cajal, completes the contributions to neurology. In medicine "Smallpox," by Schamberg, illustrated by several excellent plates, is not only timely, but valuable in its suggestions and presentation. Douglas reviews the causation and treatment of various hemorrhages, systematizing our knowledge, but without introducing any novel features. In Surgery, Deaver presents a miscellaneous article dealing with important topics, while Wathen contents himself with carefully reporting a single instance of splenectomy. The single paper on pediatrics is one on "Acute Dilatation of the Heart," by Blackader, which is characteristically readable and thorough. Casselberry, reporting on adrenalin, covers very thoroughly the field of use in this very important addition to our therapeutic resources. A notable paper by Shoemaker, "A Study of the Especial Ocular Signs Found in States of Unconsciousness," appeals rather to the physician than to the ophthalmic surgeon, but is worthy of careful study by both. In therapeutics we find several important topics ably presented. Tuffier's rather optimistic paper on "Surgical Analgesia by Injections of Cocaine into the Spinal Column," and Doléris on the "Oxytocic Effect of the Lumbar Injection of Cocaine, Particularly to Induce Labor," thoroughly cover questions which have recently become prominent. The marked results obtained by this method not only give the practitioner aid in time of need, but also warn us of danger when it is employed in general surgery. Other excellent articles are those by Jardine, Chaulford, and Broca. The volume concludes with the pronunciation and definition of some of the newer medical words, by Dorland. This is timely, in view of the rapid extension of medical nomenclature, and withal appalling in the torture to which classical Latin and Greek are put. As is apparent to the reader, this volume is well abreast of its predecessors in practical value and extent of information. R. W. W.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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The Juvenile Form of Progressive Paralysis.—Most authors understand by juvenile progressive paralysis cases in which the manifestations begin before the twentieth year. HIRSCHL (*Wiener klin. Wochenschrift*, 1901, Band xiv., p. 515), however, asserts that it would be wise to call juvenile only those cases which depend upon hereditary lues, in contradistinction to the progressive paralysis of adults, which is probably always preceded by an acquired lues.

In Krafft-Ebing's clinic 20 cases of juvenile progressive paralysis have been observed in the last ten years; 8 of these cases have been more or less fully described previously. In 17 of these instances hereditary lues was positively made out. Once a probable history was procured, while twice no evidence of lues could be obtained. In 1 of these two instances the mother probably died of general paralysis, while in the other nothing was known about the parents. In 9, or 45 per cent. of these cases, the patients were, from the onset, of feeble intellectual development, while several were also submicrocephalic; 12 of the patients were males, 8 females. The period of onset of the disease varied between the eighth and the twentieth year, figures which agree very markedly with those of Thiry's 69 cases. In most of the cases puberty had not been reached, and in those instances the patients retained during the whole course of the disease an infantile habitus. The condition began with psychical and physical symptoms, which were usually fairly well-marked; severe headache, attacks of vomiting, apoplectiform or epileptiform seizures, short lapses from consciousness, with slow recovery of speech, preceding characteristic disturbances of speech. Eye symptoms were common, as was also exaggerated knee-jerk. Irritative symptoms, tremor, and sometimes rhythmic movements of the upper extremities were common. In association with these symptoms, loss of memory

and dementia rapidly developed. These symptoms gradually increased, the physical and psychical degeneracy progressing steadily. In all cases there was but a simple dementia, never maniacal or hypochondriacal delirium. Only once, at the height of the disease, were optical hallucinations observed. In the final stages of the disease the patients were, as a rule, confined to bed as the result of the paralytic manifestations in the extremities. The duration of the disease varied greatly, averaging about three and three-quarter years. The longest cases in Krafft-Ebing's clinic were two which lasted about seven years. An autopsy was obtained in twelve cases. There was a marked diminution in weight and size of the brain; the leptomeninges were strikingly thickened; there was granular ependymitis with chronic external and internal hydrocephalus; well-marked diffuse sclerosis. Microscopically, a well-marked leptomeningitis was to be made out as well as characteristic cerebral changes.

The author believes that the following differences exist between juvenile progressive paralysis and that of adults.

1. The congenital lues as cause of the juvenile paralysis.
2. The early age at the beginning of the disease.
3. The fact that commonly individuals of weak mental development fall a prey to this form of paralysis.
4. The prodromal stadium, rich in symptoms, which, in addition, is characterized by many paroxysms.
5. The fact that at the height of the disease there is, as a rule, only simple dementia, almost never a paralytic mania or a paralytic hypochondria; that at the height of the disease there are no remissions, and that irritative symptoms as well as paroxysms are very often met with.
6. The relatively long course of the disease.
7. The discovery of a diffuse cerebral sclerosis, with marked leptomeningitis, at autopsy.

A New Case of General Paralysis, with Hereditary Syphilis.—Regis (*Arch. de Neurologie*, 1901, Band xi., 2 S., p. 449) describes a characteristic case of general paralysis developing in a young man with undoubted hereditary syphilis, at about the age of seventeen years. The course of development was that of a progressive dementia and gradually increasing enfeeblement of the intellectual faculties. The onset was gradual and the course progressive. Maniacal symptoms and hallucinations were entirely absent.

The Effects of the Growth of the Bacilli of Cholelithiasis in Bile.—During the last fifteen years numerous observers have called attention to the connection between infection of the contents of the gall-bladder with certain bacteria and the formation of gallstones. The causal relation between infection with the bacterium coli communis and the typhoid bacillus and cholelithiasis has especially been clearly shown. There is some reason to believe that the streptococcus pyogenes and the staphylococcus and, possibly, some other organisms may also play a slight part. There has been, however, considerable question as to the manner in which infection with these bacteria brings about the formation of the calculus. Italia (*La Riforma Medica*, 1901, Ann. xvii., ii., 830) has conducted a series of experi-

ments in an endeavor to elucidate this point. The organisms with which he has worked were the bacterium coli communis, the typhoid bacillus, the streptococcus pyogenes, the staphylococcus aureus, and the bacillus subtilis (extracted from water). Ox bile, filtered and sterilized, was used as a medium. This always showed a marked alkaline reaction. The bacteria were first cultivated in a medium composed of bouillon and bile, and then transplanted to pure bile. Two different classes of results were obtained, one occurring when the colon bacillus and the typhoid bacillus were used, the other with the streptococcus and staphylococcus. The typhoid and colon bacilli grow rapidly in pure bile. The medium, however, becomes neutral in from twenty to thirty days, while in from fifty to ninety days a sediment begins to be precipitated. This sediment appears earlier, and is more abundant in cultures of the typhoid bacillus than in those of the colon bacillus. If instead of keeping the bacilli in the same medium several transfers are made, it will be noted that in the third or fourth transplantation into bile the reaction after a few days changes from alkaline to acid. If these bacteria are kept in bile sixty to ninety days the reaction of culture medium also becomes sharply acid. The sediment is found to consist of crystals of carbonate of lime laden with biliary pigments and crystals of cholesterin. It would then appear to be clear that the colon bacillus and the typhoid bacillus have the property of precipitating cholesterin when cultivated in bile. This Italia believes to be due to the acid reaction which is produced. Typhoid and colon bacilli cultivated in bile become rapidly attenuated. The staphylococcus aureus and streptococcus develop poorly in bile and never change the reaction of the medium, the cultures becoming sterile in from twenty-one to thirty days. Sometimes a gradual development of the organism may be made out, the streptococcus developing better than the staphylococcus, which requires from eight to fifteen days for scanty growths. Both of these organisms give rise to a sediment in the culture, which consists in greater part of phosphates and carbonate of lime. No cholesterin is present, and the sediment is very much scantier than that obtained with typhoid and colon bacilli.

The subtilis gives a scanty sediment and grows slightly in bile without altering its reaction. If the colon bacillus be added to a culture of staphylococcus or streptococcus it develops rapidly, and a more abundant sediment is deposited. This contains much biliary pigment and carbonate of lime, and, after from ten to fifteen days, cholesterin is also found. With the precipitation of the cholesterin the reaction of the bile is always found to be acid. It appears, therefore, that the colon bacillus develops more rapidly and better in mixed infections with the staphylococcus or streptococcus than alone.

The author concludes:

1. The bacterium coli communis and the bacillus of Eberth are the specific micro-organisms of cholesterin calculi.

2. The streptococcus pyogenes and the staphylococcus aureus may rarely be the cause of cholelithiasis; and, if a calculus should occur, this would consist solely of salts of lime, as the above-mentioned micro-organisms never determine the precipitation of cholesterin

3. If the bacterium coli communis be associated with the streptococcus

and the staphylococcus, their biochemical action takes place more rapidly in the bile; the calculi in this case will be mixed—that is, composed of cholesterin, salts of lime, and biliary pigment.

The Present Position of the Bacteriology of Rheumatic Fever.—POYNTON and PAINE (*British Medical Journal*, September 21, 1901, p. 779), in discussing again the etiology of rheumatic fever, assume the position that there is no question as to the microbic origin of the disease. They are of the opinion that in the study of the bacteriology of rheumatism sufficient care has not been shown in the selection of cases. They had used only cases of rheumatic fever in childhood in their investigations. By so doing they have materially diminished the possibility of mistaking cases of arthritis deformans and gout for rheumatism, the risk of which is considerable in adults.

The writers classify the views upon the bacteriology of rheumatic fever under five groups:

1. That rheumatic fever is the result of an infection with a diplococcus. The researches of von Leyden, Triboulet, Dana, Apert, Wassermann and Malakoff, and Fitz Meyer, as well as their own, have a special bearing on this view.

2. That it is the result of an infection with a specific anaërobic bacillus. Achalme, Thiroloix, and several French observers hold that the disease is due to such a bacillus, which is of large size and remarkably variable in its morphological characters. These investigators claim to have found the organism constantly and to have produced the disease experimentally in rabbits.

3. That rheumatic fever is not due to any particular micro-organism, but is a special reaction to varied infections. The writers do not think this view is entitled to more than passing mention.

4. That simple uncomplicated rheumatism, which reacts to salicylates, is due to some unknown virus, and the complications are the results of secondary infections, usually with cocci. The writers say that if one takes the rheumatic fever of childhood as the type it is difficult to say which lesions are to be considered as complications and which as a part of the disease itself. They are of the opinion that the so-called complications are due to the same organism as are the joint manifestations.

5. That rheumatism owes its origin to staphylococcal or streptococcal infection; that it is, in fact, an attenuated pyæmia due to pyogenic cocci. This view is advanced and strongly supported by Gustav Singer.

Singer has vigorously attacked Wassermann and Fitz Meyer, who claim that the diplococcus they have isolated is specific. Poynton and Paine, who are strong advocates of the diplococcus causation of rheumatic fever, vigorously oppose Singer's theory. Singer holds that rheumatic fever cannot be produced experimentally. They state that if he would grant such a possibility, then there would be some chance of proving or disproving his view, for if it is the correct one, it should be possible to attenuate a virulent culture of the streptococcus pyogenes or staphylococcus aureus in such a way as to produce with them experimental rheumatic fever; and, on the other hand, it should be possible to raise the virulence of cultures obtained from cases

of rheumatic fever in such a way as to produce a septicæmia indistinguishable from that caused by virulent cultures of these pyogenic cocci. The writers have attempted to produce these results, but have failed.

Poynton and Paine have isolated a diplococcus in eighteen cases of rheumatic fever, and, owing to its constancy, think that they are justified in designating it as "diplococcus rheumaticus." They hold that the balance of evidence is strongly in favor of the view that rheumatic fever is due to a diplococcus infection. They hope that other investigators will eventually be able to support their view that there is a specific organism. If it be conclusively proven that there is such an organism, it may then be possible to isolate a specific toxin and eventually to manufacture a curative antitoxin.

SURGERY.

UNDER THE CHARGE OF

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The Cause of Diffuse Peritonitis Complicating Appendicitis, and its Prevention.—OCHSNER (*Journal of the American Medical Association*, June 22, 1901) states that in fatal cases of appendicitis the patient practically always dies as the result of a diffuse peritonitis. Other conditions may arise which may result in a fatal issue. There may be a septic thrombosis of the vessels in the vicinity of the appendix, or an empyema, or even pyæmia, but by far the greatest number of deaths occurs from diffuse peritonitis, and if it is possible to prevent this the mortality from appendicitis must at once fall enormously. There is the danger of the occurrence of diffuse peritonitis in the following classes of cases: 1. Gangrenous appendicitis. 2. Perforative appendicitis. 3. Cases in which the cæcal end of the lumen of the appendix is closed and the distal portion so thoroughly distended with septic material as to make its walls permeable to micro-organisms. 4. Those rare cases in which there are small abscesses in the walls of the appendix not directly connected with its lumen. 5. Those cases in which there is a septic thrombosis of some of the vessels, but not sufficient to cause gangrene. The infection of the general peritoneal cavity must occur from a disturbance on the part of the small intestines, and must be due to their peristaltic motion, so food and cathartics are absolutely contraindicated, and any portion of food which may be retained in the stomach at the beginning of the attack should be removed by gastric lavage. It may be necessary to perform gastric lavage twice, or at most three times, in order to entirely remove rem-

nants of food which may have regurgitated into the stomach from the small intestines by reason of return peristalsis. This method of treatment has proved itself of the greatest value, especially in those cases where the appendix was gangrenous or perforated, and in which there was already a beginning general peritonitis.

The nausea and vomiting will cease after one or two, or at most three, gastric irrigations. The meteorism and the pain will decrease greatly during the first twelve hours, and will almost completely disappear in twenty-four hours. The pulse becomes slower and firmer and more regular, the breathing deeper, and the patient's general appearance improves to an astonishing extent. If the temperature is high it will go below 100° F. the first twenty-four hours, and in three days it will be practically normal. The abdominal muscles will become soft as soon as the stomach contents have been removed by gastric lavage.

The author has operated in this hospital upon 565 appendicitis cases, which he has divided into three groups: 1. Those who entered the hospital suffering from diffuse peritonitis. 2. Those who entered the hospital suffering from gangrenous or perforative appendicitis. 3. Those who entered the hospital suffering from recurrent appendicitis in the interval between attacks or at the beginning of a recurrent attack when the infectious material was still confined to the appendix. Of the first class he treated eighteen cases with ten deaths, 55.5 per cent. mortality; of the second class he operated upon 179 cases, with nine deaths, 5 per cent. mortality; of the third class he operated in 368 cases, with one death, ¼ per cent. mortality. Total, 565 cases, with twenty deaths, 3.5 per cent. mortality.

These statistics contain all patients who entered the hospital suffering from appendicitis, even those who died a few hours after admission from general peritonitis.

The author states in conclusion: 1. Peristaltic motion of the small intestines is the chief means of carrying the infection from the perforated or gangrenous appendix to the other portions of the peritoneum, changing a circumscribed into a general peritonitis. 2. This can be prevented by prohibiting the use of every kind of food and cathartics by the mouth, and by employing gastric lavage in every case in which there are remnants of food in the stomach or in the intestines above the ileocæcal valve, as indicated by the presence of nausea or vomiting or meteorism. 3. The patient can be supported by the use of concentrated predigested food administered as enemata not oftener than once in four hours and not in larger quantities than four ounces at a time. 4. This form of treatment, when instituted early, will change the most violent and dangerous form of acute perforative or gangrenous appendicitis into a comparatively mild and harmless form. 5. Cases of perforative or gangrenous appendicitis, with beginning general peritonitis, can usually be carried through the acute attack safely with this method. 6. In all cases of this class gastric lavage should be practised in order to prevent the absorption of decomposing material from the alimentary canal. 7. In cases of doubtful diagnosis this form of treatment should always be employed. 8. This treatment will prevent a large proportion of the most troublesome complications and sequels of appendicitis, such as ventral hernia, fecal fistulæ, extensive adhesions, etc. 9. The patient should be permitted to

recover fully from his acute attack before an operation is performed, except in cases encountered within the first thirty-six hours after the beginning of an attack or in case of the formation of a superficial circumscribed abscess. 10. It often requires but a small amount of any kind of food to change a harmless circumscribed into a dangerous diffuse peritonitis. 11. The treatment does not protect the patient against a subsequent attack. 12. It does not contraindicate the removal of a diseased appendix before the septic material has extended beyond this organ. 13. It is indicated in all intra-abdominal conditions in which it is desirable to prevent the distribution of septic material by means of peristaltic motion. 14. The laity should be taught to stop feeding and giving cathartics to patients suffering from intra-abdominal diseases.

Hydatid Cyst of the Prostate.—BANGS (*Annals of Surgery*, May, 1901) reports the interesting case of a man, aged thirty-nine years, who presented himself for treatment for retention of urine. He was catheterized, and on the day of his admission to the hospital 162 ounces of urine were withdrawn. On emptying the bladder a large tumor was found in the hypogastrium, extending over into the left inguinal region. Examination for calculus was negative, but it showed the urethra to be ten and one-half inches in length. Rectal examination showed the prostate to be normal on the patient's right side, but the left lobe is round, much larger than the right, and its posterior and lateral margins cannot be defined from the mass in the pelvis, with which it seems to be continuous. Bimanual pressure moves the whole tumor, which is spherical in shape and somewhat elastic, but there is no sensation of fluctuation, fremitus, or ballottement. It seem to be connected with the prostate. An operation was decided upon, and a vertical incision was made in the median line from above the pubes nearly to the umbilicus. The tumor was adherent to the large iliac vessels and to the tissues of the pelvis. The tumor was aspirated, and about twenty-five ounces of fluid withdrawn. The tumor was then freely laid open, and was found to contain many secondary cysts, which were removed. As it was found impossible to remove the sac, therefore the edges of its opening were stitched with silk to the abdominal wall in the lower fifth of the incision, two drainage-tubes inserted in the sac, and the abdominal wound closed. Microscopical examination showed the presence of hooklets and many secondary cysts in the fluid evacuated from the tumor. The patient made an uninterrupted recovery. The author believes the case to be one of prostatic origin for the following reasons: 1. The length of the urethra, viz., ten and one-half inches, in a young man, aged thirty-nine years, in whom were no evidences of an inflammatory process. 2. The obstruction to urination was at the neck of the bladder and in the situation of the left lobe of the prostate, the latter being found to be asymmetrically enlarged. 3. The notable rotation toward the right of the beak of the searcher as it came in contact with the left lobe of the patient's prostate, and the left lateral deflection of the handle of the instrument to enable its distal end to curve around (so to speak) the obstruction. 4. The situation of the tumor as determined by digital examination by the rectum, as verified by two other observers; but this is admittedly inconclusive. 5. The findings at the time of the operation.

A search of the literature shows how few cases of this seat of hydatid cyst are on record. Out of twenty-three cases of hydatids of the pelvis tabulated by M. Nicaise (*Bulletin de la Société de Chirurgie*, in 1884), there are but seven which have any close relation to the prostate, and the diagnosis is so doubtful that Sir Henry Thompson does not hesitate to say: "It is doubtful if hydatid cysts have ever been met with in the prostate." He is inclined to think "that all cases of hydatid of the prostate are cases of hydatids between the bladder and the rectum, the prostate gland having been more or less absorbed by pressure from an external cyst, so that the latter came at length to occupy the seat of this organ."

Notwithstanding this ingenious argument of Sir Henry Thompson, with which he attempts to throw doubt upon the situation of the tumor, there are three cases which the author has been able to find which seem to correspond with his case and fortify him in the diagnosis which he has felt warranted in making.

Fracture of the Pelvis.—PAUL (*Annals of Surgery*, June, 1901), in reporting fifty-four cases which presented themselves for treatment at the State Hospital, Hazleton, Pa., states that fractures of the pelvis are not common, and are almost invariably due to external violence delivered directly or indirectly upon some portion of the affected structure. Any of the pelvic bones may be broken, and multiple fractures are not unusual. According to some authorities, the pubes is the part most often affected; according to others, the ilium; at any rate fractures of the ischium and acetabulum are uncommon, and of the sacrum and coccyx rare. Compound fractures are rare, but the author's table includes five cases. Pelvic fractures are often comminuted. In dislocations of the symphysis or fractures of the pubes rupture of the urethra is very common; next in frequency comes rupture of the bladder, which may be extraperitoneal or intraperitoneal. Urinary extravasation accompanies the latter, and is often associated with the former, but retention is the usual sequel of urethral laceration. Cases complicated by rupture of the rectum are rare; the author is able to report only one. Rupture of the great vessels in the pelvis is probably more common than is suspected, the symptoms being ascribed to "shock." Contusion or rupture of the kidney is frequently associated with fracture of the ilium. In two cases luxation of the hip complicated the pelvic fracture. Deformity is never well marked. In some cases it is easy to elicit abnormal mobility and crepitus; but in others local tenderness is all that can be found. The prognosis in all cases should be guarded. Simple uncomplicated cases of fractured pubis require rest in the recumbent position, with pressure so exerted as to keep the fragments in apposition. If the pubic fracture be complicated with laceration of the urethra, a catheter should be tied in this canal for several days. Should it be impossible to introduce the catheter an external urethrotomy should be performed. If the bladder is ruptured into the peritoneal cavity, a laparotomy should be done immediately and the rent sutured. If the rupture is extraperitoneal, the bladder should be constantly drained, urinary antiseptics, such as salol and boric acid, administered, and early free incisions made to relieve extravasations. Some cases, especially those in which other portions of the body are injured, will be

attended with so much shock as to absolutely contraindicate all operative procedures.

In some cases of fractured ilium the fragments remain in apposition themselves, in others reduction cannot be maintained. Should the broken fragment become united in its abnormal position, loss of function or apparent deformity rarely result. Unless a fragment of the ilium has a tendency to be displaced outward, which is unusual, broad bands of adhesive plaster or muslin should *not* be drawn around the pelvis. Should the upper portion of the acetabular rim be broken off, the head of the femur should be restored to its cavity and extension applied. It is difficult to prevent forward displacement of the lower end of the sacrum where this bone is broken. It is desirable, for various reasons, to avoid inserting packing or canulas into the rectum to prevent this displacement. Malgaigne's hooks, or a modification thereof, applied to the back of the sacrum might prove useful in these cases. Fracture of the coccyx requires usually only rest for its treatment, and should coccygodynia result excision is demanded. The author's table of fifty-four cases includes only one fracture in a woman. Of these cases twenty-seven died, two were improved, and twenty-five were cured.

THERAPEUTICS.

UNDER THE CHARGE OF

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Use of Drugs in Pulmonary Tuberculosis.—DR. W. R. HUGGARD says that there is a middle course to pursue between that advocated by those who overestimate the power of drugs in the treatment of phthisis and those who underestimate the same. The first point made clear is that up to the present no specific has been found for tuberculosis, and also that there is no drug that is useful in all cases. Nevertheless, there are various drugs that favorably influence the course of some varieties of the disease, always provided that they are well tolerated by the patient, and that they do not cause any disturbance of appetite, digestion, or assimilation. The main objects to be attained are: 1. Improving the general health, as, for example, by arsenic, quinine, strychnine, lime, phosphorus preparations. 2. Increasing the local resistance of the affected tissues, as by the use of creosote and its derivatives, salicylic preparations, and counter-irritants. 3. Modifying the quantity or character of the secretions, as by employment of the balsams, the terebinthinates, the essential oils, morphine and apomorphine, and inhalations,

especially of formaldehyde. 4. Controlling systems that react prejudicially on the patient. Among these are: (*a*) Digestive ailments; (*b*) excessive or needless cough, which shakes and exhausts the patient, causes fever, or prevents sleep; (*c*) scanty expectoration and retention of secretions; (*d*) fever, which spoils appetite and prostrates the patient. 5. Removing complications, such as syphilis. The most important point in the treatment is to consider the state of the digestive system. Temperature is the next most important guide to treatment. Pyrexia will in a large proportion of cases yield to absolute rest, bodily and mental, combined with life in the fresh air. In many cases, however, a return to a normal level of temperature can be hastened by the administration of small doses of phenacetin—one to three grains—in combination with quinine and salol and sometimes arsenic. Phenacetin seems to have rather a tonic than a prejudicial effect on the heart and on the general health, and may without hesitation be continued for months, if need be. Next to pyrexia as a guide to treatment should be placed a marked tendency to hæmoptysis. The author thinks that creosote and guaiacol increase the liability to pulmonary hemorrhage—as well, in fact, as to pulmonary inflammation—and should be avoided where such a disposition is present. On the contrary, the lime salts, the terebinthinates, and the balsams have a somewhat restraining influence, as has also morphine in minute doses. Summing up, the author says that the treatment is determined, in the first instance, by the digestive system, by the general health, and by the state of nutrition. If digestion is bad the only drugs indicated are such as will restore it to a normal condition. If digestion is good, the general condition satisfactory, and the patient improving, he refrains from using drugs unless some definite indication is present. Among the most useful indications for drugs are persistent afternoon pyrexia in spite of absolute rest out-of-doors, a tendency to recurrent febrile attacks or to slight inflammatory attacks. This tendency is usually combined with impaired nutrition and with a low state of general health. In these conditions arsenic, strychnine, quinine, and salol are among the most useful tonics. Active softening, excessive cough, overabundant expectoration, and, more rarely, scanty expectoration, expectoration of extremely purulent or nummular character, require attention; and the drugs useful for these conditions generally seem to him to have a favorable influence on the course of the disease in addition to the temporary relief from the discomfort they may afford. Among such drugs are formaldehyde vapor, creosote and its derivatives, except guaiacol carbonate, which the author has found inert, terpin hydrate, oil of cinnamon, myrtol, the balsams, and the lime salts. In sluggish chronic softening, counter-irritation by iodine or by small flying blisters is of the greatest use.—*Medical Press and Circular*, 1901, No. 3250, p. 184.

Atropine and Intestinal Obstruction.—A number of observations have recently been made bearing on the use of large doses of atropine for the treatment of intestinal obstruction. Drs. Aronheim, F. H. Pritchard, and H Gebele all discuss this subject. The patient of Aronheim had great pain in the left hypogastrium, and other symptoms of intestinal obstruction developed. One-twentieth of a grain of atropine was given, and was followed by an easing of the pain in one hour, and later by the passage of a

large, semi-solid mass of feces. Dilatation of the pupil and dryness of the throat were marked. Pritchard describes a case with similar symptoms in a man, aged sixty-two years. Hot applications and enemata were of no value, and one-thirty-third of a grain of atropine was given in two doses, four hours apart. This was followed by the passage of large movements and the recovery of a large gallstone. Gebele discusses the subject from a critical point of view, and draws less favorable conclusions. He subdivides the cases and states that in the dynamic cases alone is the treatment of avail. The truly mechanical cases are less amenable.—*Münchener med. Wochenschrift*, 1901, No. 33, pp. 1313 *et seq.*

Tuberculosis Treated by Urea.—Dr. Arthur H. Buch, following the lines indicated by Harper, already noted in these abstracts, reports some favorable results from the use of urea in both localized and general tuberculosis. The histories of seven patients are given; these include those suffering from tuberculosis of ankle-bones and elbow-bones, with lupus of nostrils, lupus of face, two cases; tuberculous adenitis, two cases; tuberculous joint, and tuberculous cold abscesses. All of these patients were reported as cured.—*Medical Press and Circular*, 1901, No. 3249, p. 163.

Chlorosis Treated by Copper.—Dr. E. Liegeois follows out the teaching of Mendini, his predecessor for fourteen years, in employing copper salts in chlorosis, amenorrhœa, and cervical lymphadenitis. The acetophosphate is the form preferred, and in many of the cases under observation for the past twenty-five years marked improvement in the blood condition has resulted.—*Journal des Praticiens*, 1901, vol. xv. p. 225.

Antiseptics and Irritants and Skin Action.—Dr. W. d'Este Emery, in a research whose object was to determine the relative irritating power of the various antiseptic and other drugs which are usually applied to the skin, arrives at some important conclusions. The methods by which all fallacies should be excluded and quantitative results obtained gave some difficulty because of the various inherent factors, such as the relative irritant potency of the substance, the strength of the solution, the reflex activity of the nervous system, the area of the skin in which the substance acts, and the length of time during which it is applied. The method of experiment was to use a decapitated frog and to determine the amount of irritant which would cause the well-known skin reflex in this animal. As yet the experiments are incomplete, and a comparatively small number of substances have been investigated, but the following conclusions are of interest: Lysol was the most irritating substance worked with, and represents one hundred on the scale; izal was sixty-six, carbolic acid thirty-eight, mercuric bichloride thirty-one, formalin eight, and alcohol six-hundredths. Thus a lotion of lysol (1 per cent.) will cause as much irritation in the skin of a frog as a lotion of carbolic acid of thrice the strength.—*British Medical Journal*, 1901, No. 2115, p. 112.

Eruptions Due to Orthoform.—Dr. W. Dubreuile reports two separate varieties of eruption consequent to the use of orthoform. These are an

erythema, alone or in combination with vesicles, and a rare type of gangrenous eruption. The former is a transitory affair, and may arise when the powder is dusted on the healthy skin. The second type has been observed while varicose ulcers have been under treatment. This type is accompanied by much pain, but is amenable to treatment.—*La Presse Médicale*, 1901, No. 40, p. 340.

Chronic Fluorine Poisoning.—Dr. F. Schwyzer reports in commendable detail an interesting case of chronic fluorine poisoning. The patient was a drinker of large quantities of beer, and it is presumed that the fluorine came from the fluorides of the glass. The subject is a complex one, and is handled in an able manner.—*New York Medical Journal*, 1901, vol. lxxiv., p. 1.

Fusel-oil Poisoning.—Dr. Thomas B. Futcher, in an interesting study of this condition, concludes that from the study of the effects of the administration of fusel oil to dogs, and from the observations on two cases of fusel-oil poisoning, the following conclusions may be drawn: 1. Fusel oil, when administered to animals, causes the elimination in the urine of combined glycuronic acid, which reduces alkaline copper and bismuth solutions, and acts as a levorotator to polarized rays. 2. When taken by man it acts as a profound intoxicant, causing unconsciousness of several hours' duration. 3. In one of the cases it was followed by symptoms of hemiplegia. 4. In certain cases, as in one cited, fusel oil is a profound blood destroyer and causes methæmoglobinuria. 5. In both cases it caused transitory nephritis. 6. In both patients a glycosuria lasting two or three days was produced. 7. In the two cases there was fairly conclusive evidence that combined glycuronic acid (*gepaarte glukoronsäure*) was present in addition to glucose.—*American Medicine*, 1901, vol. ii., p. 210.

Critical Review of the Literature of Toxicology.—Dr. A. Heffter, of Bern, contributes an important critical résumé of the toxicological literature appearing during the years 1897–1900. To those interested this is an important summary.—*Schmidt's Jahrbücher*, 1901, vol. cclxx., p. 151.

OBSTETRICS.

UNDER THE CHARGE OF

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Cæsarean Section for Eclampsia.—In the *Monatsschrift für Geburtshülfe und Gynäkologie*, 1901, Band xiv., Heft 2) Sippel discusses the question of Cæsarean section for eclampsia. Recognizing virulent toxæmia to be the cause of eclampsia, he draws attention to the apparent coincidence between

the cessation of pregnancy and the cessation of the eclamptic paroxysms. Reasoning from this, he believes rapid delivery is indicated in these cases. Where the cervix is undilated and labor has not begun, he considers Cæsarean section indicated. He believes it to be a less irritating traumatism than incision and dilatation of the cervix. He admits that many cases are unsuitable, and quotes Hillmann's statistics of 40 cases with 19 maternal recoveries, and of 41 children 23 saved.

He reports the case of a primipara, aged seventeen years, who was taken with severe eclampsia five weeks before term. On examination the child was living, in the second position, the smaller fontanelle directed backward and to the right and the cervix undilated. The birth-canal was small and ill developed. To give mother and child the best chance for recovery Cæsarean section was performed. Gauze was placed in the cervix as a drain. The child, although asphyxiated, was resuscitated, and survived.

The mother improved after the operation and the secretion of urine became re-established. She had intestinal hemorrhage, from which she died on the seventh day. An autopsy showed an ulcer of the duodenum as the site of the hemorrhage. The kidneys showed fatty degeneration of the epithelia, and both ureters were dilated.

It is evidently unfair in this case to ascribe death to the results of the operation. The tendency which all eclamptic patients have to bleed from the mucous membranes must be kept in mind in considering the question of the duodenal ulcer.

The Operative Treatment of Collections of Pus in the Uterine Appendages.—MANDL and BÜRGER (*Archiv für Gynäkologie*, 1901, Band lxiv., Heft 1) contribute an interesting paper upon this subject which is of especial interest to obstetricians in view of the treatment of pelvic abscess following puerperal septic infection. The material for this paper included 273 cases in Schauta's practice. The operations adopted in these cases consisted of the removal by laparotomy of pus-containing tubes, the removal of the tubes and uterus by laparotomy, incision and drainage of purulent collections through the abdominal wall, the removal of the uterus and both tubes through the vagina, the removal of a diseased tube through the vagina, with the preservation of the uterus and the other tube, and the opening and emptying of a tubal abscess through the vagina.

The writers report their experience with these various methods with that of other operators. The question of drainage is fully discussed, and also the permanent effects secured by various methods of operation. The writers draw attention to the fact that disease of the Fallopian tubes is rarely unilateral, and that operation which removes one tube only must often be supplemented by a second operation, removing the remaining tube. The writers state a very important general principle which is now recognized by surgeons, namely, that in the presence of fever operation should be deferred if possible. If the patient is growing worse vaginal incision and drainage should be performed. When the patient is convalescent and the pus has become practically sterile, radical operations should be undertaken.

The writers conclude that if possible operations for the relief of abscesses in the uterine appendages should be done through the vagina to avoid

infecting the peritoneum. When both Fallopian tubes are involved in suppuration they do not advise removal of both tubes by abdominal section. They believe that the operation is often followed by the unfavorable complications attending abdominal section, and that a permanent satisfactory result is rarely obtained. In cases where the disease is limited to one Fallopian tube, and the surrounding tissues are healthy, such tube may be removed to advantage by abdominal section. It is often necessary in these cases to make a preliminary puncture to ascertain the condition of the pus before operating. Where removal through the vagina offers great technical difficulties abdominal section may be chosen. The removal of both suppurating tubes and diseased tissues is best performed by vaginal section. The permanent results are better and the patient escapes the unfavorable complications of abdominal section. The same ground of reasoning applies to the removal of one Fallopian tube through the vagina as stated with reference to the abdominal operation. Only when the pus contained is known to be sterile is the operation advisable. Vaginal incision cannot be considered as an operation of cardinal value, but as a means for diagnosis and of meeting threatened complications. In exceptional cases abdominal incision and drainage of collections of pus may become necessary.

Pus in Abdominal Operations.—ROBB (*American Journal of Obstetrics*, August, 1901) reports 72 operations for collections of pus in the pelvis. He agrees with others that during an acute attack operation should not be undertaken, but that the patient should be kept quiet on her back in bed and heat applied to the abdomen. A vaginal douche of a gallon of warm 1 per cent. solution of carbolic acid or saturated boric-acid solution is given twice daily. The patient is nourished by nutrient injections and anodynes, and heart stimulants are given if necessary. Should no improvement take place, and a mass be discernible in the pelvis, it is punctured through the vagina and the sac irrigated and packed with sterile gauze.

The micro-organisms found in his cases were staphylococci, streptococci, gonococci, and the colon bacillus. The tubercle bacillus was occasionally seen. The streptococcus was much more deadly than the others, and produced fatal peritonitis. Streptococci may be present when organs are adherent, even when no pus can be demonstrated and cover-slip preparations at the time of operation may fail to show the presence of organisms. The operator should allow a day or two to elapse after operation before performing further operations, to avoid the risk of infection. Gauze packing, the free use of sterile salt solution, the use of the cautery upon pedicles, and leaving warm sterile salt solution in the abdomen, are all means of value in preventing the development of acute infection.

In 49 of the 72 pregnancy had occurred at some time. Infection after labor, or miscarriage, or gonorrhœal infection was the most usual cause of disease in these cases. In 26 the appendix was removed. The operation most frequently performed was the removal of both tubes and ovaries. Hysterectomy was done but once; dilatation and curetting in one-third of the cases; in 9 cases drainage was carried through the cul-de-sac alone; in 2 cases through the abdominal wall alone, and in 1 case through both. In 2 cases in which infection occurred after the original operation the abdomen

was opened and drained. Suppuration of the abdominal wall occurred in 12 cases, or 18.7 per cent.

Two patients died, a mortality of 2.77 per cent. In 1 general septic infection following pelvic abscesses was present, and cultures from the lungs showed a pure streptococcus infection. In the second case streptococci appeared in culture tubes two days after operation, and the diplococcus of pneumonia and the bacillus coli communis.

GYNECOLOGY.

UNDER THE CHARGE OF

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Calcification of Fibroids.—GUIBÉ (*Annales de Gynécologie et d'Obstetrique*, July, 1901) reviews the pathology of this condition, which he believes is due to some unknown circulatory disturbance.

While there are no characteristic symptoms, the patient often complains of a sensation of weight, with severe lancinating pains radiating through the loins and downward to the perineum and lower limbs. These are probably due partly to irritation of the peritoneum and partly to stretching of the nerves and uterine ligaments. Pressure symptoms are frequent, especially œdema of the lower limbs. Expulsion of calcified fragments is the only positive evidence of the presence of an intra-uterine growth, though a fetid discharge was present in 25 per cent. of the reported cases.

The prognosis is usually favorable, though deaths from peritonitis and intestinal obstruction have been noted.

The positive recognition of the presence of calcareous degeneration of a fibroid is only possible when a hard body can be felt inside of the uterus or fragments are discharged. Occasionally the characteristic hard tumor can be detected by abdominal palpation. Operation is indicated to relieve marked pressure symptoms and in the presence of peritonitis and intestinal obstruction, the abdominal route being preferred, except in cases of intra-uterine growths.

Ligation of the Uterine Arteries in Cases of Fibroid.—KAKONSHKIN (*La Gynécologie*, June 15, 1901) reports seven cases and analyzes eighty-five others. The immediate results of the operation were quite successful, only one case being a failure. Hemorrhage and pains were relieved, and the tumor usually diminished in size. The best results were obtained in cases of interstitial growths, especially in old patients.

Unfortunately, the results are not permanent on account of the establishment of the collateral circulation, hence ligation of the vessels should be

reserved for cases in which the patient is unable to sustain a radical operation or refuses to allow one.

Vaginal Enterocele.—GOUILLOND (*Annales de Gynécologie et d'Obstétrique*, July, 1901) reports a case of posterior vaginal enterocele, which was cured by three operations—shortening of the round ligaments, anterior colporrhaphy, and resection of the posterior cul-de-sac, with posterior colporrhaphy. The writer believes that many cases of posterior vaginal enterocele have been mistaken for extensive rectocele, and operated upon within proper reduction of the hernia and resection of the sac.

Epithelium of the Fallopian Tube.—VOINOT (*Thèse de Paris; La Gynécologie*, June 15, 1901), from careful studies of the anatomy of the tube, arrives at the conclusion that ciliated epithelium is present both before puberty and after the menopause. Before puberty cilia are found most often at the pavilion, less often at the ampulla, and often at the ampulla and isthmus, especially between the folds. After the menopause they are found only at the ampulla. During the period of sexual activity cilia are most numerous, but are disposed in groups, being found most constantly in the depressions between the folds.

Surgical Treatment of Pelvic Adhesions.—OIT (*La Gynécologie*, June 16, 1901) discusses the question of intrapelvic adhesions and the painful symptoms to which they give rise. He believes that it is not sufficient merely to divide or to resect them, since they always tend to re-form.

The operation is performed most easily by making an incision through the posterior vaginal fornix. The adhesions are located, divided, and the raw surfaces are sutured in a direction at right angles to their long axes. The writer reports a considerable number of successful cases. It is important to select them carefully, avoiding those in which there is a possibility of there being suppurative foci.

Decidua Ovarii.—LINDENTHAL (*Centralblatt für Gynäkologie*, 1901, No. 28) affirms that in the second month of pregnancy spaces are seen in the tunica albuginea due to local œdema, which later are filled with cells bearing a close resemblance to those of the decidua. At the seventh month they begin to undergo retrograde changes, some atrophy, but the majority become vacuolated, and are transformed into permanent hyaline tissue. The writer was always able to demonstrate the presence of such islands of hyaline tissue in the ovaries of women who had borne children, but never in those of nulliparæ.

The condition described as superficial papilloma he believes are simply projections of such tissue through the albuginea. Ingrowths of germinal epithelium are cut off by the development of these islands of hyaline tissue, and cavities are formed lined with epithelium, the latter subsequently forming ovarian cystomata, as is well known. Hence the inference that these decidual formations which develop in the ovary during pregnancy are to be regarded as an important etiological factor in the genesis of simple ovarian cysts.

OPHTHALMOLOGY.

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A Case of Bilateral Retinal Detachment During Labor Associated with Albuminuria; Complete Recovery.—HANN and KNAGGS (*The Lancet*, May, 18, 1901) report the case of a woman, aged twenty-one years, who became suddenly affected with blindness after she had been in labor twenty-four hours. There was œdema of the hands, feet, and legs for about three weeks previous. For three hours the pains continued with great intensity, then the patient fell into a state of semi-coma, with stertorous breathing, from which she would be roused by the pains. Labor was terminated by forceps. Three days later an ophthalmoscopic examination made under atropine showed large detachments of the retina filling up the lower third of each globe. They floated up readily on movements of the eyes, and had all the usual characters of detached retinæ. Four weeks later the retinal detachments were no longer to be seen, though there was perhaps some slight appearance of persistence at the very margin. The vision was: right, 6/18; left, 6/12, partly. The fields showed definite contraction in the upper hemisphere. The urine contained a ring of albumin and a trace of sugar, no casts. Two weeks later the vision was: right and left, 6/9 partly. All traces of detachment had disappeared. Given the general tendency to œdema depending upon the renal disease, the mechanical condition affecting the circulation induced by labor was probably quite sufficient to account for the detachment. There was no evidence of renal retinitis. Ophthalmic surgeons must be familiar with cases in which a history is given of eclamptic attacks and of blindness following confinement and ending in recovery. These cases are rarely investigated at the most interesting period, and when they are subsequently seen there is probably nothing found to explain what the exact condition was. Possibly retinal detachment or sudden œdema of the choroid and retina may explain some of them.

The Sometimes Successful Treatment of Cases of Apparently Incurable Blindness.—The age of miracles is not yet passed. Witness the results reported by DR. CHARLES BELL TAYLOR in a clinical lecture with the above title, in *The Lancet*, April 27, 1901. The cases comprise patients who have recovered after many years of apparently hopeless blindness, others who have suffered from and under treatment have recovered from the effects of neuroretinitis, iritis with diffuse scleroderma, hyalitis, detached retina, atrophy of the optic disk, and the results of destructive inflammatory and

degenerative changes. "As to remedies outside the usual routine, I should certainly place electricity first; mercury in large doses alone, or in combination with other drugs, second; derivatives, vascular, nervous, or both, third; and lastly, persevering treatment on these lines with such operative procedures as may be indicated." Reports of cases are given; here is one: A man, aged thirty-five years, was sent to be taught a trade in a blind asylum; he had lost his sight from severe optic neuritis; the disks were white and blurred, and he could not tell day from night; he had been treated with the usual remedies, and the case seemed hopeless. Powerful galvanic currents were applied daily and sometimes twice a day. Result: he was able to resume his former occupation of house painter, and he can now (twelve years later) read the newspaper in a bad light, and distant vision is perfect.

The following cases illustrate the statement that mercury, "this much-abused remedy alone or in combination is simply marvellous:" A girl, aged fifteen years, was quite blind from iritis and parenchymatous keratitis; no iris or pupil visible, the eyes were simple opaque masses; mercury in large doses for four years; perfect recovery. A myopic peer (sic) had suffered from iridochoroiditis with effusion into the vitreous, no reflex from either eye, but detachment of the retina was suspected by the writer. Within a month's treatment by galvanism, large doses of mercury, massage, pilocarpine, and derivatives he recovered excellent sight, as good as he had ever had, and which continued until his death many years later. The lecturer had three additional almost identical cases, and many others which differed only in small details. Among the former a "distinguished journalist" and a "well-known merchant."

Finally, a number of cases are reported to illustrate the good effect of operation combined with treatment on above lines. These cases yield in no respect to those previously detailed. It is difficult to select from *embaras de richesses*. The case of the "son of a member of Parliament" is a type.

The Action of Becquerel and Rontgen Rays Upon the Eye.—F. HIMSTEDT and W. A. NAGEL (*Annal. d. Physik.*, Fol. iv., p. 537). Preparations of radium emit, according to Becquerel, so-called invisible rays. Giesel had already found that a combination of radium enclosed in paper impervious to light and laid upon the eye excites a faintly luminous sensation, filling the entire eye. Himstedt and Nagel now show that this sensation is due to fluorescence of the solid and fluid media brought about by the radium rays. The sensation is very like that occasioned by the ultra-violet rays, whose action as exciters of fluorescence has been previously shown. These observers, like Dorn and Brandes, also obtained localized light effects from the Röntgen rays, but unlike those occasioned by the Becquerel rays, which take the form of a diffuse faint light. As a matter of fact, the Röntgen rays effect no fluorescence of the refractive media. More rarely, and thus far unexplained, is the fact that the Röntgen as well as the Becquerel rays give an impression of greatest intensity in that part of the eye exactly corresponding to the point of excitation; thus, if the rays enter the eye from above, the superior part of the retina is excited, whereas ordinarily the sensation is projected to the opposite extremity of the diameter, passing through the nodal point, for rays coming above the projection point is below.

The prepared eye of the frog shows, as is well known, a current which changes upon entrance of light from rest to action current of activity. The ultra-violet rays, as already proven, occasion the same phenomena, and, as Himstedt and Nagel now show, the same is true of the Röntgen rays.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF
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The Finer Changes in the Peripheral Portion of Divided Nerves.—MURAWIEFF (*Ziegler's Beiträge*, 1901, vol. xxix. p. 102) found the formol-methyl method of great service in the study of both normal and pathological nerve fibres. This method was devised several years ago by Rossolimo and Murawieff.

The fresh nerve is placed in a 4 per cent. formalin solution, where it should remain not less than two or three days. It is then transferred to 95 per cent. alcohol for twenty-four hours, but never longer than forty-eight hours.

Tease out a small piece of nerve and stain for ten to fifteen minutes in a one-half per cent. aqueous solution of methylene blue, which is heated until the first bubbles appear. Cool the solution and move the tissue into aniline oil for one-half second (aniline oil, 1 part; 95 per cent. alcohol, 9 parts), then into 95 per cent. alcohol for one-half second. Clear in cajuput oil. Place the bits of nerve on a slide and add a drop of Canada balsam. Tease the nerve fibres thoroughly and cover the preparation with a cover-slip. If one wishes to make a cross-section of the nerve, then, after hardening in the 4 per cent. formalin, place it in 95 per cent. alcohol twenty-four hours; then in absolute alcohol twenty-four hours. Embed in celloidin. Stain in the manner already described.

When a normal nerve is treated according to this method the medullary sheath is seen to be studded with small, dark blue granules, which are distributed uniformly. They are approximately of the same size, except near the nodes of Ranvier, where there are larger granules or small masses. The granules are arranged in two concentric rings; one close to the axis-cylinder, the other near the periphery of the medullary sheath. Although Murawieff made a careful study of the granules, he was not able to determine whether they exist in the myelin sheath of the lining nerve fibre or whether they are the result of post-mortem changes.

The author studied the changes in the peripheral portions of nerves after division. His observations were made on rabbits and guinea-pigs. The absorption of the myelin forms the essential part of the "degeneration" of the nerve fibre. It is due to the cessation or very great reduction of the synthetic processes in the myelin. Chemical changes appear later. They exhibit marked variations and cannot in any case play the chief part in the process of "degeneration."

The nuclei of Schwann's sheath and the protoplasm surrounding them show great vital activity. The nuclei increase in size and in number, and the protoplasm increases markedly. This rapid growth is explained partly by the increased supply of food material to the cells in part, probably in consequence of the disturbance of the tissue equilibrium and a certain irritation produced by the degenerating myelin. The nuclei multiply and the protoplasm increases markedly. The growth of the latter becomes so excessive that nourishment fails, and it undergoes fatty degeneration and then necrosis.—[J. H. P.]

The Etiology of Cancer. V. LEYDEN (*Zeitsch. f. klin. Med.*, 1901, vol. xliii., p. 1) examined the ascitic fluid from a girl, aged sixteen years, with a malignant ovarian tumor. The fluid was cloudy, and in it were many peculiar cells which were motile. These cells protruded long protoplasmic processes, and fat granules in the cells moved into these processes. The cells did not possess powers of locomotion. V. Leyden believed that these cells were protozoa and were accidentally present, but since has found similar cells in ascitic and pleuritic fluids in cases of cancer, and now believes that they are characteristic of cancer.

Cultures from bits of fresh cancer were made upon a culture medium of fuscus crispus, and in six to eight days well-developed amœbæ were found.

In cabbage occurs a peculiar growth (Kohlhernie) analogous to "human cancer," due to the action of a parasite. In sections of this growth inclusions are found in the cells like those seen in cancer cells.

V. Leyden examined sections of cancer of the breast and axillary lymph nodes, and saw in cell protoplasm spherical bodies, with a dark staining centre, surrounded by a clear space which does not stain. He thinks that these bodies resemble protozoa. He believes they do not arise from the nucleus, and are not degenerations, but are amœbæ. He saw in one cell an appearance like that of a sporocyst.

V. Leyden has not seen the various intermediate stages of development. He believes they are not yeasts, for yeasts grow between and not in cells.

[V. Leyden describes the familiar cancer-cell inclusions. He claims that these inclusions are protozoa purely on morphological grounds. He has not been able to cultivate the organism or produce tumors by inoculating them. He does not prove the identity of his "motile" cells with the cancer-cell inclusions. He has no evidence that his "motile" cells are protozoa or the cause of the epithelial-cell growth. His article is valueless as evidence of the parasitic nature of cancer]. [E. H. N.]

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VOL. CXXII., No. 7. DECEMBER, 1901. No. 357.

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Published Monthly.

EDITED BY FRANCIS R. PACKARD, M.D.

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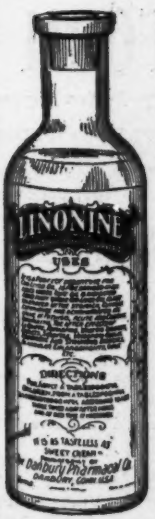

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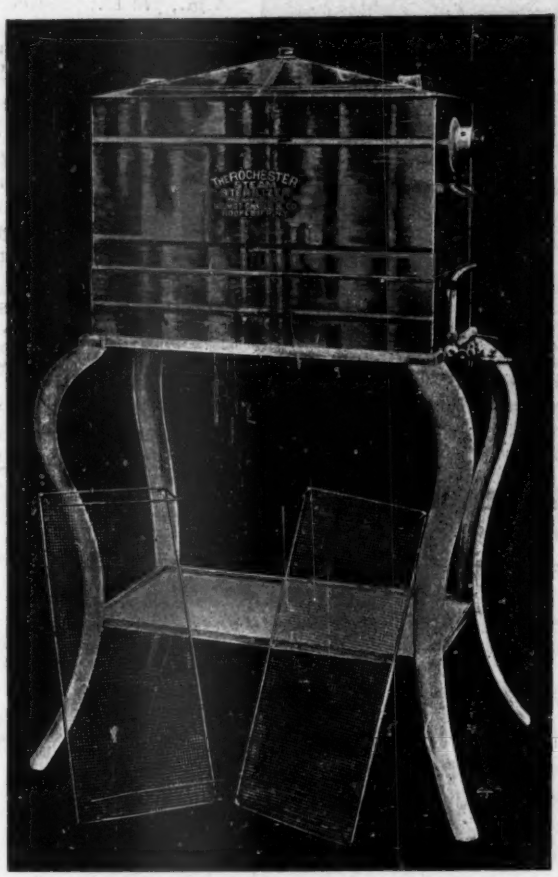

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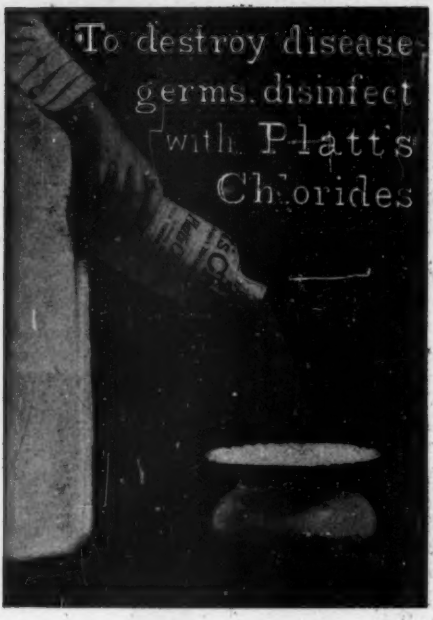

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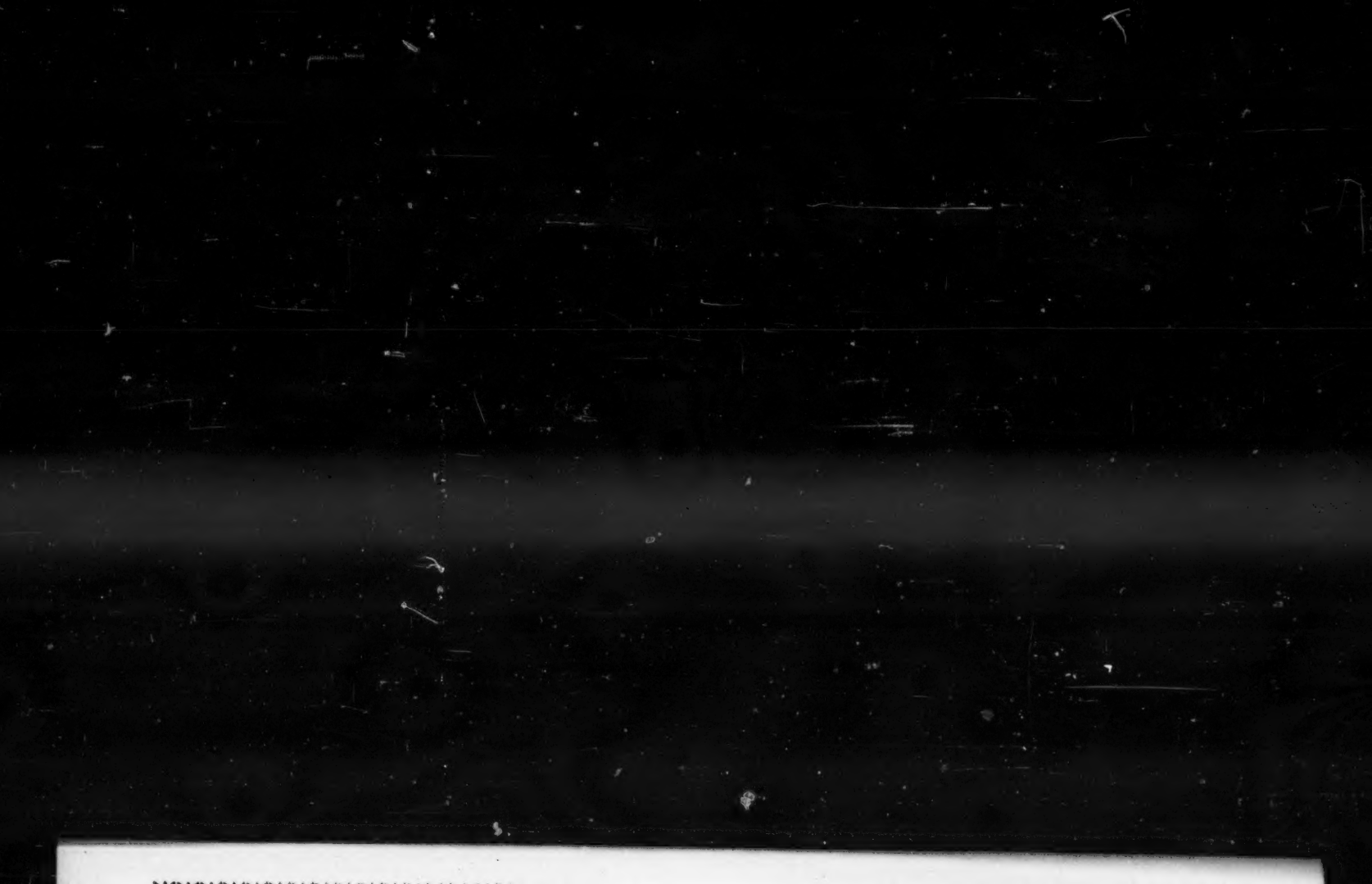

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